A STUDY ON LIPID ABNORMALITIES IN HYPERTENSIVE PATIENTS

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BONAFIDE CERTIFICATE

This is to certify that **" A STUDY ON LIPID ABNORMALITIES IN HYPERTENSIVE PATIENTS"** is bonafide work done by **Dr. N.RAVI SHANKAR** post graduate student, Department of General Medicine, Kilpauk Medical College, Chennai-10, under my guidance and supervision in partial fulfillment of regulations of **The Tamilnadu Dr.M.G.R.Medical University** for the award of **M.D.Degree Branch I (General Medicine)** during the academic period from May 2007 to March 2010.

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CONTENTS

HYPERTENSION AND LIPID ABNORMALITIES

INTRODUCTION

Earliest references about Hypertension are seen in Chinese books written about 2600 B.C. Until prior to twentieth century there were no clinical instrument for measuring blood pressure non invasively. However the presence of high blood pressure had long been recognized by the degree of " hardness of arteries ". Even though the association of high blood pressure and major cardio- vascular events like stroke and cardiac failure were recognized by ancient physicians like Hippocrates and Galen, the real breakthrough came in the early twentieth century after the introduction of non-invasive BP monitoring with sphygmomanometer by Riva Roci[1] and Koratkoff. Further insights into variation of BP in humans under the effect of exercise, stress, emotion and the complication of longstanding hypertension were made possible. Today hypertension has been recognized as the most common cardiovascular disorder[2]. It is the leading cause of morbidity and mortality in both developing and developed countries[3].

Hypertension is one of the ten leading reported causes of death with about 4% of such death due to hypertensive complications [4] . The risk features that have been associated with hypertension include

increased salt intake, Diabetes mellitus, Cigarette smoking, elevated serum lipids, sedentary lifestyle , diet rich in saturated fats , genetic factors and stress [5]

Though the association of hypertension & dyslipidemia is common and been proved by various studies beyond doubt, the reason for this co-occurrence has not been probed out yet . Three possible mechanisms are proposed for this but none have been proven.

1. Dyslipidemia can increase the incidence of Hypertension.

2. Hypertension can increase the incidence of Dyslipidemia.

3. There may be a common factor which cause increased incidence of both.

This study is conducted to assess the abnormalities in plasma lipid profile of hypertensive patients and to determine the factors influencing it.

AIM OF STUDY

- To study the prevalence and pattern of lipid profile abnormalities in newly diagnosed hypertensive patients.
- To study the influence of various clinical, demographic , social and socioeconomic parameters on lipid profile abnormalities in hypertensive patients.

REVIEW OF LITERATURE

HYPERTENSION

EPIDEMIOLOGY AND RISKS

Approximately 1 billion people worldwide are affected by hypertension [6]. There are however important differences in prevalence between population and ethnic groups [7]. The Framingham Heart study has estimated that individuals normotensive at age 55 years have 90 % lifetime risk of developing hypertension [8]. The fourth National Health and Nutrition examination survey (NHANES IV) showed an overall prevalence of hypertension 28.7 % of United States population . The prevalence varied from 7.2% in those aged 18- 39 years to 65.4 % in those aged 6 years and older. It was greater in women (30 .1 %) than in men (27.1 %)

Hypertension represents a potent risk factor for cardiovascular, peripheral vascular and renal diseases [9,10,11,12,13]. The relationship is strong, continuous and gradual. A 36 year follow up from Framingham Heart study showed that hypertension is associated with 2 to 4 fold increase in the risk for the development of coronary heart disease, cardiac failure and overall cardiovascular events in both men and women.

The Framingham study also predicts a 1.7 fold increase in the risk of stroke per S.D increment in women and 1.9 fold increase in risk per S.D increment in men at age 6O.It also showed peripheral arterial disease risk to increase dramatically by 3 fold in males and 4 fold in females.

The relation of increasing BP levels to incidence of ESRD is well determined. MRFIT showed relative risk of ESRD to be 1.7 in men for every 16 mm Hg increase in SBP. Baseline SBP of > 14O mm Hg were associated with a 5-6 fold greater risk for ESRD compared with SBP below 117 mm Hg .

PATHOPHYSIOLOGY OF HYPERTENSION

ENVIRONMENTAL AND PSYCOSOCIAL STRESS

Observation of people who move from stable rural traditional societies to unstable urban environment showed increase in BP over the lifespan [14] . Westernized people with low socio economic status have higher BP [15]. Type A or coronary prone behavior pattern is associated with increased BP and risk of Coronary heart disease[16]. Anger arousing experiments have long been known to raise BP and increase vasoconstriction[17,18]. Other psychological factors related to increase in BP are power motivation (desire to dominate others),

depression[19,20], hopelessness and pessimism . The effect of persistent hopelessness was equivalent to that of smoking 2.5 packs of cigarettes daily [21,22] . The stress buffers like social support, aerobic exercises and stress reduction interventions like bio feedback , relaxation training and cognitive behavioral therapies have been shown to lower B.P. with mixed success [23].

RENIN-ANGIOTENSIN SYSTEM (RAS)

In humans, rennin is coded for by a single gene in chromosome-1. It is secreted by juxta- glomerular cells in response to decrease perfusion. The renin converts angiotensinogen to angiotensin I which is converted to angiotensin II by ACE. Angiotensin II is thought to be responsible for most of the physiologic and pathophysiologic effects of RAS. Evidences are accumulating that ACE polymorphism due to insertion or deletion may be responsible at least a part to the pathology of essential hypertension[24,25,26].

ALDOSTERONE AND MINERALOCORTICOIDS.

Apart from the physiological role in retaining Na + and excreting K + in epithelial cell, Aldosterone has other less explored functions like vasoconstriction through its direct action on vascular smooth

muscle, in brain to stimulate salt appetite and direct action on cardiac myocytes which may contribute to essential hypertension.

INSULIN RESISTANCE AND HYPERINSULINEMIA.

Though the relation between hyperinsulinemia and hypertension is well established the mechanisms by which it rises the BP remains doubtful and complex. The possible mechanisms include.

1. Insulin mediated salt retention [27,28].
2. Insulin activation of sympathetic nervous system [29,30].
3. Proliferative effects of insulin[31].
4. Non enzymatic glycation of interstitial tissues in arterial walls [32].
5. Insulin induced endothelial dysfunction leading to arterial stiffness.

REMODELLING OF RESISTANT ARTERIES IN HYPERTENSION.

Abnormalities of endothelial cells, smooth muscle cells, adhesion molecules and extra cellular matrix in vasculature may contribute to structural, mechanical and functional changes that reduce lumen diameter of small arteries (400 – 100 micron).There are two types of remodeling.

1. Eutrophic remodeling in which the cross sectional area of lumen is maintained

2. Hypertrophic remodeling in which the cross sectional area of lumen is increased.

Of these the Eutrophic remodeling occurs in essential hypertension.

ENDOTHELIUM IN HYPERTENSION

The two major endothelium derived vasoactive substances are Nitric oxide (NO) and Endothelin (ET).Nitric oxide is released from endothelium in response to shear stress and exerts vasodilating and antiproliferative effects on smooth muscle cells. It also inhibits the thrombocyte aggregation and leucocyte adhesion. The whole body Nitric oxide production in patients with essential hypertension is diminished under basal conditions[33,34]. Oxidative stress plays an important role in the pathogenesis of hypertension by oxidation of Nitric oxide to peroxynimine by superoxide anion thereby effectively reducing the bioavailability of Nitric oxide [35,36].

Endothelin exerts its major vascular effects, vasoconstriction and cell proliferation through ET A receptors on vascular smooth muscle cells. In contrast ET B receptor mediate vasodilation via release of NO and prostacyclin. Apart from the effects on blood vessels it also causes sodium retention and Renin release in kidneys , Aldosterone release from

adrenals and hypertrophy and fibrosis of heart musculature.These effects may contribute to essential hypertension in human[37,38,39].

NATRIURETIC AND VASODILATORY PEPTIDES

The natriuretic peptides including ANP , BNP , CNP , DNP and urodilantin performs multiple functions like natriuresis , vasodilation , anti- proliferative effects , vascular remodeling and modulation of nor adrenergic and RAAS . In addition to these, the vasodilatory peptides like calcitonin gene related peptide (CGRP) , substance P and Adrenomedullin regulates cardiovascular function in normal state and in hypertension.

THE KALLIKREIN – KININ SYSTEM

The kininogenic enzymes act on kininogen to form kinins. The kinins act via two receptors . The B1 receptor mediates inflammation , pain and fibrosis, the B2 receptor mediates the depressor , natriuretic , antitrophic and fibrinolytic functions via mediators like Eicasanoids , EDHF (endothelium derived hyperpolarizing factors) , NO, t-PA etc.

CONCEPT OF VASCULAR STIFFENING

A decreased distensibility of aorta and other large arteries or the loss of the windkessel function is known to be a cause for hypertension.

This vascular stiffening develops from complex interaction between structural and celluar elements of vessel wall . The structural components include 2 prominent scaffolding proteins , collagen and elastin . Dysregulation of the balance between the production and degradation of collagen and decreased production of elastin which contribute to vascular stiffness [40]. These vascular alterations are influenced by intrinsic factors [41] like hemodynamic forces and extrinsic factors like hormones,salt,glucose and lipids [42].

In addition to the structural components , cellular components also play a role in the pathogenesis of vascular stiffening . They are signals from endothelial cells like NO and Endothelin -1 and the vascular smooth muscle tone The Vascular smooth muscle tone is modified by mechanostimulation and by paracrine mediators like Angiotensin II , ET -1 , oxidative stress and nitric oxide[43]

DEFININIG HYPERTENSION

The multiple risk factor intervention tria (MRFIT) demonstrated a continous and graded influence of both systolic and diastolic blood pressure of coronary heart disease mortality extending down to systolic blood pressures of 120 mm Hg. Therefore from an epidemiologic perspective ,there is no obvious level of blood pressure that

defines hypertension. Clinically "Hypertension may be defined as that level of Blood pressure at which the institution of therapy reduces Blood pressure related morbidity and mortality". The JNC VII classifies hypertension as follows

	SBP (mm Hg)		DBP (mm Hg)
Normal	<120	and	<80
Pre HT	120-139	Or	80-89
Stage 1	140-159	Or	90-99
Stage 2	>/ 160	Or	>/100
ISH	>/140	And	<90

CLINICAL DISORDERS OF HYPERTENSION

Depending upon the methods of patient ascertainment, 80-95% of hypertensive patients are diagnosed to have "Essential Hypertension (also called as Primary or Idiopathic Hypertension) . In the remaining 5-20 % , a specific underlying disorder causing the elevation of blood pressure can be identified. These group is called " SECONDARY HYPERTENSION".Essential Hypertension tends to be familial and is

likely to be the consequences of an interaction between environmental and genetic factors.

PATHOLOGIC CONSEQUENCES OF HYPERTENSION

Hypertension is a risk factor for atherosclerosis . It is an independent predisposing factor for heart failure,coronary artery disease, stroke, renal disease and peripheral arterial disease.

HEART

Hypertensive heart disease is the result of structural and functional adaptations of heart leading to left ventricular hypertrophy, diastoloic dysfunction, congestive heart failure, coronary heart disease, microvascular disease and cardiac arrythmias.

BRAIN

Hypertension is an important risk factor for brain infarction and haemorrhage . Hypertension is also associated with an impaired cognition in aging population . In malignant hypertension there is loss of cerebral auto regulation leading to hyperperfusion causing encephalopathy which may progress to stupor , coma, seizures and death within hours if left untreated.

KIDNEY

Hypertension is an important risk factor for renal injury and ESRD. The atherosclerotic hypertension related vascular lesion in kidney primary affect the pre glomerular arterioles , resulting in ischemic changes in glomeruli and post glomerular structures . The injury may also be due to direct damage to the glomerular capillaries due to hyperperfusion . The pathology progress to glomerulosclerosis and eventually to gradual atrophy of tubules. The renal lesion associated with malignant hypertension consists of fibrinoid necrosis of afferent arterioles and sometimes the necrosis of glomerular tuft.

PERIPHERAL ARTERIES

In addition to contributing to the pathogenesis of hypertension, blood vessels may be a target organ for hypertension. Intermittent claudication is the classical symptom of peripheral artery disease.Retinopathy is also common.

PATIENT APPROACH & EVALUATION

The initial assessment include a complete history to confirm a diagnosis and to screen for cardiovascular risk factors to rule out

secondary causes of hypertension . Most of the hypertensive patients have no specific symptoms referable to elevated BP . Headache generally occurs only in patients with severe hypertension . Characteristically a hypertensive headache occurs in the morning and is localized to occipital region . Other non specific symptoms related to elevated BP include dizziness, palpitation , easy fatiguability and impotence.

MEASUREMENT OF BP

Before taking the BP measurement , the individual should be seated quietly for 5 min in a private quiet setting with comfortable room temperature. The centre of the cuff should be at the heart level and the width of the bladder cuff should be equal to atleast 40% of the arm circumference. The rate of deflation of the cuff is 2 mm Hg /sec. systolic blood pressure is the first of atleast two regular " tapping " Korotkoff sounds and diastolic blood pressure is the point at which the last regular Korotkoff sound is heard . In current practice, a diagnosis of hypertension is generally based on seated office measurement.

Currently available ambulatory monitors are fully automated , use oscillometric technique and typically are programmed to take BP every 15-30 mins. Ambulatory monitoring of BP is not routinely used and

reserved for patients in whom hypertension is suspected.TheJNC VII has also recommended ambulatory monitoring for treatment of resistant, symptomatic hypotension , autonomic failure and episodic hypertension.

PHYSICAL EXAMINATION

Body habitus include weight and height should be noted. At the initial examination BP should be measured in both arms and preferably in supine, sitting and standing positions. In hypertension diagnosed before the age of 30 years, lower limb BP should be checked. All the peripheral pulses should be examined for rate, rhythm , volume and character. Heart rate should be recorded . Neck should be palpated for thyroid gland. Fundoscopic examination should be done. Auscultation for bruit over carotids,femorals and renal arteries is done. cardiac auscultation for loud S2 and S4 Gallop is done. Palpation of the precordium is done for a heaving apical impulse.

LABORATORY TESTING

Basic laboratory testing for initial evalution

RENAL	ELECTROLYTES	METABOLIC	OTHERS
Urine microscopy Urine albumin BUN Ser.creatinine	Ser.sodiun Ser.potassium Ser.calcium	FBS Lipid profile	Hematocrit ECG

The aim of anti - hypertensive management by various means is to bring down and to maintain the BP below 140/90 mmHg.In diabetic patients the BP goal is 130/80 mmHg and in nephropathy patients it is125/75 mmHg.

DYSLIPIDEMIA

Dyslipidemia is the most prevalent and important modifiable risk factor for atherosclerosis . Proper treatment reduces the risk for cardiac death, non fatal MI, stroke, and peripheral vascular disease by 25 – 50 %. Despite these benefits only 20 % of adults meet national guidelines for cholesterol control.

Dyslipidemia is defined as abnormal lipid status. Common lipid abnormalities include elevated total cholesterol, LDL cholesterol,

lipoprotein (a) and triglycerides , low level of HDL cholesterol and a preponderance of small density LDL particles. These abnormalities occur alone or in combination.

PREVALANCE, RISK AND SCREENING

Approximately 50% of US adults have an elevated total cholesterol. In a vast majority of the patients with atherosclerotic vascular disease there is some form of dyslipidemia even though their total cholesterol is normal. Elevated total Cholesterol , LDL c and low HDL c levels are major modifiable risk factors for coronary heart disease and other forms of atherosclerotic vascular disease. ATP III recommends a routiene lipoprotein analysis for all adults aged 20 years. screening should be repeated every 5 years . Since dyslipidemia is an asymptomatic condition, early recognition and treatment improves prognosis.

EFFECT OF THERAPY

Several large randomized , placebo controlled trials of statin therapy have shown reduction in cardio vascular morbidity and mortality. A meta-analysis of more than 330 interventional trials has shown that for every 1 % total cholesterol, mortality is reduced by 1.5 % and for each 1 % reduction in LDL c and for each 1 % increase in HDL c, the risk

for cardiovascular events is reduced by 2 % and 3 % respectively [44].A number of studies with serial angiography also show that the increase in stenosis was 1-3 % less per year in aggressively treated patients than in placebo. The lower level of LDL c maximum benefit extends to as low as 50 -70 mg/dl for high risk patients.

LDL CHOLESTEROL GOALS

The LDLc goals are individualized depending on their CHD risk

MAJOR RISK FACTORS THAT MODIFY LDL GOALS [45]

The first step in estimating the LDL c goal is counting the number of risk factors

Cigarette smoking	Age (male $\geq$ 45 yrs ; women $\geq$ 55 yrs)
Hypertension	Obesity (BMI $\geq$ 30 kg/m2)
HDL c (<40 mg/dl)	Physical inactivity
Diabetes mellitus	Atherogenic diet
Family history of premature CHD	Emerging risks

The next step in calculation of the 10 year risk of coronary heart disease using the Framingham heart study risk score. Based on these,

individuals are classified in 5 groups and their LDL c goals are set.

VERY HIGH RISK GROUP- LDL c goal < 70 mg %[46]

10 year risk for major coronary event > 20 %

Established CHD

CHD equivalents

Diabetes mellitus

Multiple cardiovascular risk factors

HIGH RISK - LDL c goal < 100 mg % ,optional goal < 70 mg %

Coronary heart disease

Coronary heart disease risk equivalents

10 year risk > 20 %

≥ 2 risk factors

MODERATELY HIGH RISK -LDLc goal < 130mg%,optional< 100 mg %

10 year risk 10- 20 %

≥ 2 risk factors

MODERATE RISK -LDL c goal < 130 mg %

10 year risk < 10 %

≥ 2 risk factors

LOWER RISK- LDL c goal < 160 mg %

0-1 risk factors

NON HDL CHOLESTEROL [47]

Non – HDL c is calculated by subtracting HDL c from TC.The primary goal of therapy for persons with dyslipedimia is LDL-c lowering . A secondary goal of non -HDL c is set by ATP III guidelines.

High / Very high risk - < 130 mg/dl.(optional < 100 mg / dl)

Moderately high risk - < 160 mg/dl.(optional < 130 mg / dl)

Moderate risk - < 160 mg / dl

Low risk - < 190 mg / dl

TREATMENT OF DYSLIPIDEMIA

THEREPEUTIC LIFE STYLE CHANGE [48,49,50].

This includes Diet , physical activity , weight reduction and smoking cessation . In many individuals this could reduce total cholesterol by < 10%.

DRUGS

DRUGS	MECHANISM	LIPID LOWERING
Statins	HMG CoA reductase inhibitors	LDL c : 18 – 55 % HDL c : 5 – 15 % TG : 7 – 30 %
Ezetimibe	Decrease cholesterol absorption	LDL c : 18 – 20 % HDL c : 1 – 5 % TG : 5 – 11 %
Niacin	Decrease production and release of VLDL	LDL c : 5 – 25 % HDL c : 15 – 35 % TG : 20 – 50 %
Bile acid sequesterants	Prevents enterohepatic circulation of BA	LDL c : 15 – 30 % HDL c : 3 – 5 % TG : unaffected
Fibrates	Increase LPL activity	LDL c : 5 – 20 % HDL c : 10 – 35 % TG : 20 – 50 %
Omega 3 fatty acids	DHA and EPA reduce VLDL production	LDL c : 44 % incr HDL c : 9 % TG : 45 %

DYSLIPIDEMIA AND HYPERTENSION -

THE METABOLIC SYNDROME

It consists of a constellation of metabolic abnormalities that confer increased risk of cardio vascular disease and diabetes mellitus.

NCEP: ATP III, 2001 CRITERIA FOR METABOLIC SYNDROME

Three or more of the following :

1.central obesity:waist circumference for male>102 cm and female >88 cm.

2.Hypertriglyceridemia: Triglycerides ≥150 mg/dl or on medication

3.Low HDL c : < 40 mg/dl for males and < 50 mg/dl for females or on medication.

4.Hypertension: Blood pressure ≥ 130 mm Hg Systolic or ≥ 85 mmHg diastolic

5.Fasting plasma glucose: >= 100 mg /dl or specific medication

Detailed analysis of the components of Metabolic syndrome have been done and the influence of one component on the other have been found out. Various cross sectional studies have suggested a link between abnormal lipids and hypertension. A few studies have

prospectively examined the relationship between plasma lipids and further development of hypertension. Small trials have looked at the effect of lipid lowering on BP.

ROLE OF LIPIDS IN THE PATHOGENESIS OF HYPERTENSION.

Dyslipidemia, causes endothelial damage and the loss of physiological vasomotor activity that may become manifested as increased blood pressure. Atherogenic lipid abnormalities cause endothelial dysfunction [51]. A dysfunctional endothelium through impaired nitric oxide production and activity and alterations in Endothelin – 1 and Endothelin –A & B receptor expression[52], cannot respond to intra – vascular conditions to dilate as needed. This vaso- dysregulation eventually leads to increased resting B.P.

Nickenig and Harrison[53,54] have linked lipids and hypertension via a mechanism of angiotensin-I overexpression.Lipid abnormalities and insulin resistance have been associated with sympathetic hyperfunction[55].

CLINICAL TRIALS IN PATIENTS WITH HT & DYSLIPIDEMIA

ASCOT-LLA TRIAL[56]

(Anglo –Scandinavian cardiac outcome Trial-Lipid lowering arm)

10,305 men and women aged 40-79 years with hypertension and ≥ 3 other risk factors and elevated cholesterol were randomized to Atorvastatin 10 mg or placebo. The primary end point : coronary heart disease, death or non fatal MI.

The Atorvastatin arm of the trial was stopped prematurely at 3.3 years due to a significant 36% reduction in the primary end point. Benefits were apparent in the first year . Atorvastatin also reduced fatal or non fatal stroke by 27%, total cardiovascular events by 21 % and total coronary events by 29 % . At 1 year Atorvastatin reduced total and LDL cholesterol by 24% and 35% respectively.

ALLHAT – LLT [57]

(Anti hypertensive and lipid lowering treatment to prevent heart attack trial)

10,355 men and women aged ≥55 years with stage 1 and 2 hypertension , ≥ 2 additional coronary heart disease risk factor and LDL c 120 -189 mg/dl (100 – 129 mg/dl in patients with CHD) were randomized to Pravastatin 40 mg/dl or usual care . Mean follow up for 4.8 years.

The results showed Pravastatin did not significantly reduce all cause mortality (primary end point) or coronary Heart disease events (

relative risk 0.91 p=0.16). Lack of benefit is attributed to substantial (30 %) use of statins in the placebo group , resulting in a modest 17 % differential in total cholesterol between Pravastatin and usual case groups.

PHYSICIAN HEALTH STUDY [5]

It was a randomized double blind placebo contolled trial of aspirin and beta – carotene in the primary prevention of cardiovascular disease and cancer. The study consisted of 22 , 071 male physicians between the age 40 – 84. From this 3110 men free of hypertension, CHD, and cancer were selected and TC and HDL c were measured and non – HDL and TC /HDL C ratio were calculated. Over mean follow up of 14.1 years , 1019 men developed hypertension . In cox proportional hazards model adjusted for lifestyle and clinical risk factors, men in the highest quintile of TC , non – HDL c and TC /HDL-c ratio had increased risks of developing hypertension of 23%, 39% and 54% respectively , compared with the participants in lowest quintile.

Furthermore mean in highest quintile of HDL-C had 32 % decreased risk of developing hypertension compared with those in the lowest quintile . These prospective cohort data suggest that dyslipidemia may lead to subsequent development of hypertension.

PLASMA LIPID PROFILE IN HYPERTENSIVE NIGERIANS [59]

Lipid profile studied in 150 hypertensive patients aged 30 – 59 and 30 years and socio economic status matched normotensive controls using standard laboratory techniques . Of the hypertensive patients 54% were females and 46% were males.Hypertensive patients heve significantly higher lipid profiles except for HDLc which did not showed any difference in the two groups.

LIPID PROFILE OF HYPERTENSIVE PATIENTS IN SPAIN [60]

In " San Cecilio " university hospital,Granada,Spain 50 patients were studied. They were divided into two groups.27 recently diagnosed hypertensive patients and 23 healthy controls of similar mean age (45 years) and BMI. Biochemical determinations were performed by automated techniques.Student's T test and Welch's test was used for statistical analysis .Hypertensive patients showed higher level of creatinine , uric acid , total cholesterol,triglycerides and chlorides than controls.

LIPID PROFILE OF HYPERTENSIVE PATIENTS IN BANGLADESH [61]

This prospective study carried out in department of bio chemistry and molecular biology , university of Rajshahi , Bangaladesh. 60

human subjects of age ranging from 33 – 60 years were studied . Among these 40 were hypertensive and 20 were normotensive . Their lipid profile results were collected and analyzed statistically. The total cholesterol (241.25 vs 182.14) , triglycerides (184.77 vs 142.73) and LDL c (154.32 vs 105.73) were significantly higher and HDL c (32.91 vs 42.88) was significantly low among hypertensive patients.

MULTICENTRIC HYPERTENSIVE POPULATION STUDY IN UAE [62]

Study conducted in 162 hypertensive and 112 normotensive matched for age , gender , & ethnicity to determine ET-1 , NO, lipid profile. Levels of VLDL and TG were significantly higher ($P < 0.01$) in hypertensive. In contrast total cholesterol ($P < 0.01$) and LDL c ($P < 0.001$) were lower among hypertensive. ET-1 and NO were significantly higher in hypertensive.

MATERIALS AND METHODS

Patients who are diagnosed as hypertensive in medical OP department, ward and hypertension clinic of Kilpauk medical college govt hospital were taken.Study population included patients belonging to urban and semi urban city of Chennai. Study period is from January 2009 to October 2009.

The Study design is Cross sectional study with cases and controls.

The study group included

- Newly detected hypertensive patients of age group between 31-75 yrs.
- Control group is non-hypertensive patients of same age group who attended medical OPD for minor illnesses.

Following group were excluded from the study

- Patients who are already known hypertensive and on drugs.
- Patients with secondary hypertension.
- Newly diagnosed hypertensive patients with one or more complications like CVA, IHD, nephropathy and retinopathy at presentation.
- Hypertensive patients who are alcoholic.
- Hypertensive diabetic patients.

The participants were explained about the study and informed consent was obtained. Then they were interviewed and analyzed for exclusion criteria. Cases which meet both the inclusion criteria and did not have any of the exclusion criteria were selected to participate in the study.

107 such cases 60 controls were included in the study. Detailed history regarding patient's education, occupation , family income, daily physical activities, smoking, alcohol intake and family history of hypertension were asked.The socio economic status of the patient was determined using " Modified (2007) Kuppuswamy scale ". Participants with daily physical activity of ≤ 2 MET (Metabolic Equivalent of Task) were considered as sedentary. Those who smoke ≥ 5 cigarettes/day were considered as smokers.

Waist circumference was measured in a horizontal plane at the level of the narrowest part between the costal margin and the iliac crest . Hip circumference was measured at the largest protrusion of the buttock with thin clothes without compressing the skin. Body mass index (BMI) was calculated using the formula

$$BMI = \text{Weight in kg} / (\text{Height in meter})^2$$

The waist hip ratio was also calculated. Participants with BMI ≥ 25 kg/m², waist circumference ≥ 88 cm in females and ≥ 102 cm in

males and WHR ≥ 0.85 in females and ≥ 0.90 in males were considered obese.Blood pressure was measured in the right arm in patient in sitting posture. It was measured after 30 min of rest and arm supported at heart level . They were also abstained from smoking and ingestion of caffeine within the previous 6 hrs . Two such readings were taken at least 24 hrs apart and the average of the two was taken.

5 ml of venous blood sample after an over night 12 hrs fasting was collected for investigation. A 2 hrs postprandial sample was also collected. The TC, TG and HDL c were determined using enzymatic calorimetric method . The LDL-c and VLDL-c were estimated using Friedeward formula.

$$\text{LDL-c} = \text{TC} - (\text{HDL} + \text{VLDL})$$

Participants with fasting blood sugar values from 100-125 mg/dl and postprandial blood sugar values from 140-199 mg/dl were considered to be pre-diabetic. Those who had TC ≥ 200 mg/dl or TG ≥ 150 mg/dl or LDL c ≥ 130 mg/dl or HDL c< 40 mg/dl were considered as dyslipidemic . Unpaired, double tailed student's T test was used to find out the significance of difference between the two means.The significance of difference in the percentage of dyslipidemia among each group was analysed using chi-square test.

RESULTS AND ANALYSIS

- 107 patients with newly diagnosed hypertension from hypertension clinic were included in the study group.
- 60 non-hypertensive persons of same age group were included in the study as control.

To study the prevalence of dyslipidemia the hypertensive patients were compared with the normotensive group.

To study the influence of various parameters on lipid profile, patients from the hypertensive group only are selected. Patients who are positive for the parameters to be tested act as cases and those who are negative act as controls.

With the available data two type of analysis were done.

- The mean values of Total cholesterol and other sub-groups of cholesterol are calculated for cases and controls and their differences were analyzed for statistical significance . The statistical analysis is done using unpaired – T test, double tailed with unequal variance.
- The percentage of dyslipidemia prevalence for among cases and controls are calculated and compared. The percentage prevalence is analyzed for statistical significance using Chi-square test.

SUMMARY OF STATISTICS FOR CONTINUOUS VARIABLES

S.NO	ITEMS	MEAN	MEDIAN	RANGE
1	AGE	55.9159	58	30-75
2	HEIGHT	157.907	158	138-174
3	WEIGHT	67.4579	65	50-97
4	BMI	27.2507	26.67276	19.8-40.4
5	WAIST-CIRCUM	94.7664	95	78-116
6	WHR	0.915415	0.918967	0.76-1.07
7	SBP	162.710	160	130-200
8	DBP	93.2897	90	10-140
9	FBS	95.7196	96	72-123
10	PPBS	126.121	126	93-190
11	TC	196.804	191	150-283
12	TG	197.312	180	58-495
13	LDL	119.153	117	64-190
14	HDL	37.935	38	24-53
15	VLDL	40.0617	36.8	12-99

TABLE-1: **MEAN LIPID VALUES: CASES VS CONTROLS**

LIPID	' N '		MEAN	SE	' P '
TC	**CASES**	**107**	**197**	**2.67**	**< 0.0001**
	CONTROL	**60**	**166**	**2.77**	**SIGNIFICANT**
TG	**CASES**	**107**	**197**	**7.03**	**< 0.0001**
	CONTROL	**60**	**120**	**5.40**	**SIGNIFICANT**
LDL	**CASES**	**107**	**119**	**2.45**	**< 0.0001**
	CONTROL	**60**	**98.4**	**2.74**	**SIGNIFICANT**
HDL	**CASES**	**107**	**37.9**	**0.63**	**< 0.0001**
	CONTROL	**60**	**42.3**	**0.98**	**SIGNIFICANT**

INTERPRETATION

The total cholesterol, triglycerides, LDL-c and HDL-c are significantly higher in hypertensive patients (cases) when compared with non-hypertensive patients (control).

TABLE-2: **PERCENTAGE OF DYSLIPIDEMIA:**

CASES VS CONTROLS

LIPID	' N '		PERCENT	' P '
TC	**CASES**	**107**	**43.92**	< **0.0001** **SIGNIFICANT**
	CONTROL	**60**	**5**	
TG	**CASES**	**107**	**84.11**	< **0.0001** **SIGNIFICANT**
	CONTROL	**60**	**20**	
LDL	**CASES**	**107**	**28.03**	**0.0003** **SIGNIFICANT**
	CONTROL	**60**	**5**	
HDL	**CASES**	**107**	**53.27**	**0.005** **SIGNIFICANT**
	CONTROL	**60**	**30**	

INTERPRETATION:

In our study, dyslipidemia is defined as TC ≥ 200 mg/dl, TG ≥ 150 mg/dl,LDL ≥ 130 mg/dl and HDL < 40 mg/dl. Cases have significantly higher percentage of dyslipidemics when compared with control.

LIPID	' N '		MEAN	SE	' P '
TC	**YRS (31-45)**	**25**	**189**	**4.08**	**0.049**
	YRS (61-75)	**47**	**203**	**4.38**	**SIGNIFICANT**
TG	**YRS (31-45)**	**25**	**182**	**12.88**	**0.12**
	YRS (46-60)	**35**	**217**	**16.73**	**IN-SIGNIFICANT**

TABLE-3: MEAN LIPID VALUES IN DIFFERENT AGE-GROUPS

LDL	**YRS (46-60)**	**35**	**111**	**4.77**	**0.0072**
	YRS (61-75)	**47**	**127**	**3.44**	**SIGNIFICANT**
LIPID	' N '		PERCENTAGE		' P '
	YRS (31-45)	**25**	**20.00**		**0.0151**
TC	**YRS (31-45)**	**25**	**38.4**	**1.23**	**0.60**
HDL	**YRS (61-75)**	**47**	**53.19**		**SIGNIFICANT**
	YRS (61-75)	**47**	**37.6**	**0.97**	**IN-SIGNIFICANT**
TG	**YRS (46-60)**	**35**	**82.85**		**0.9637**

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TERPRETATION

Three age groups were formed among the hypertensive patients. Group-I: 31-45yrs, group-II: 46-60yrs and group-III: 61-75yrs. The total cholesterol is significantly high among hypertensive patients of group-III when compared with group-I.

TABLE-4: PERCENTAGE OF DYSLIPIDEMIA IN AGE-GROUPS

	YRS (61-75)	**47**	**85.10**	**IN-SIGNIFICANT**
LDL	**YRS (31-45)**	**25**	**20.00**	**0.2459**
	YRS (61-75)	**47**	**36.17**	**IN-SIGNIFICANT**
HDL	**YRS (31-45)**	**25**	**52.00**	**0.9861**
	YRS (46-60)	**35**	**54.28**	**IN-SIGNIFICANT**

INTERPRETATION

Significant percentage of dyslipidemics is present in group-III with respect to total cholesterol and triglycerides when compared with groups with lowest lipid values.

TABLE-5: MEAN LIPID VALUES: MALES VS FEMALES

LIPID		'N'	MEAN	SE	'P'
TC	**MALE**	**58**	**198**	**3.62**	**0.50**
	FEMALE	**49**	**195**	**3.96**	**IN-SIGNIFICANT**
TG	**MALE**	**58**	**201**	**8.29**	**0.52**
	FEMALE	**49**	**192**	**11.88**	**IN-SIGNIFICANT**
LDL	**MALE**	**58**	**121**	**3.11**	**0.31**
	FEMALE	**46**	**116**	**3.89**	**IN-SIGNIFICANT**
HDL	**MALE**	**58**	**36.4**	**0.78**	**0.0084**
	FEMALE	**49**	**39.7**	**0.96**	**SIGNIFICANT**

INTERPRETATION

Hypertensive females have significantly higher HDL levels when compared with hypertensive males.

TABLE-6: PERCENTAGE OF DYSLIPIDEMIA: MALES VS FEMALES.

TC	**MALE**	**58**	**50.00**	**0.2448**
	FEMALE	**49**	**38.77**	**IN-SIGNIFICANT**
LIPID	N	MEAN	SE	SIG
TG	**MALE**	**58**	**87.93**	**0.2398**
	FEMALE	**49**	**79.59**	**IN-SIGNIFICANT**
LDL	**MALE**	**58**	**34.48**	**0.1063**
	FEMALE	**49**	**20.40**	**IN-SIGNIFICANT**
HDL	**MALE**	**58**	**63.79**	**0.0176**
	FEMALE	**49**	**40.08**	**SIGNIFICANT**

INTERPRETATION

Significant percentages of female hypertensive patients have HDL values > 40 mg/dl

TABLE-7: MEAN LIPID VALUES: SMOKERS VS NON-SMOKERS

TC	**SMOKER**	**22**	**215**	**6.49**	**0.00014 SIGNIFICANT**
	NON-SMOKER	**36**	**188**	**3.32**	
TG	**SMOKER**	**22**	**233**	**16.98**	**0.0019 SIGNIFICANT**
	NON-SMOKER	**36**	**182**	**6.77**	
LDL	**SMOKER**	**22**	**129**	**6.89**	**0.07327 IN-SIGNIFICANT**
	NON-SMOKER	**36**	**117**	**2.55**	
HDL	**SMOKER**	**22**	**39.3**	**1.33**	**0.00304 SIGNIFICANT**
	NON-SMOKER	**36**	**34.7**	**0.85**	

IINTERPRETATION

Hypertensive smokers have significantly higher TC, TG and HDL values when compared with hypertensive non-smoker males.

TABLE-8: PERCENTAGE OF DYSLIPIDEMIA

SMOKERS VS NON-SMOKERS.

LIPID	' N '	MEAN	SE	' P '

LIPID	' N '		PERCENTAGE	' P '
TC	**SMOKER**	**22**	**69.56**	**0.0085**
	NON-SMOKE	**36**	**34.28**	**SIGNIFICANT**
TG	**SMOKER**	**22**	**95.65**	**0.2236**
	NON-SMOKE	**36**	**85.71**	**IN-SIGNIFICANT**
LDL	**SMOKER**	**22**	**39.13**	**0.4018**
	NON-SMOKE	**36**	**28.57**	**IN-SIGNIFICANT**
HDL	**SMOKER**	**22**	**56.52**	**0.35**
	NON-SMOKE	**36**	**68.57**	**IN-SIGNIFICANT**

INTERPRETATION

Hypertensive smokers have significantly high percentage of patients with TC in dyslipidemic range (TC ≥ 200 mg/dl) when compared with hypertensive non-smokers.

TABLE-9: MEAN LIPID VALUES: BODY MASS INDEX (BMI)

TC	**OBESE**	**74**	**205**	**3.12**	**< 0.0001**
LIPID	**NON-OBESE**	**33**	**177** PERCENTAGE	**3.09**	**SIGNIFICANT**
TG	**OBESE**	**74**	**209**	**9.42**	**0.011**
	NON-OBESE	**33**	**171**	**6.72**	**SIGNIFICANT**
LDL	**OBESE**	**74**	**124**	**3.24**	**0.0035**
	NON-OBESE	**33**	**109**	**2.40**	**SIGNIFICANT**
HDL	**OBESE**	**74**	**39.6**	**0.68**	**< 0.0001**
	NON-OBESE	**33**	**34.1**	**1.08**	**SIGNIFICANT**

INTERPRETATION

Hypertensive patients with BMI ≥ 25 kg/m² are considered obese and < 25 kg/m² as non-obese. Obese patients showed significantly higher values of all lipid parameters.

TABLE-10: PERCENTAGE OF DYSLIPIDEMIA: BODY-MASS INDEX

TC	**OBESE**	**74**	**58.10**	**< 0.0001**
	NON-OBESE	**33**	**12.12**	**SIGNIFICANT**
LIPID	' N '		MEAN SE	' P '
TG	**OBESE**	**74**	**87.83**	**0.1149**
	NON-OBESE	**33**	**75.75**	**IN-SIGNIFICANT**
LDL	**OBESE**	**74**	**36.48**	**0.0036**
	NON-OBESE	**33**	**9.09**	**SIGNIFICANT**
HDL	**OBESE**	**74**	**44.59**	**0.007**
	NON-OBESE	**33**	**72.72**	**SIGNIFICANT**
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INTERPRETATION

Hypertensive patients with BMI ≥ 25 kg/m² have significantly higher percentage of TC, LDL and HDL in dyslipidemic range when compared with hypertensive patients with BMI < 25 kg/m².

TABLE-11: MEAN LIPID VALUES: WAIST - CIRCUMFERENCE

TC	**OBESE**	**48**	**208**	**3.95**	**0.00015**
	NON-OBESI	**59**	**188**	**3.20**	**SIGNIFICANT**
TG	**OBESE**	**48**	**215**	**12.48**	**0.024**
	NON-OBESI	**59**	**183**	**7.30**	**SIGNIFICANT**
LDL	**OBESE**	**48**	**126**	**4.34**	**0.019**
	NON-OBESI	**59**	**114**	**2.55**	**SIGNIFICANT**
HDL	**OBESE**	**48**	**39.2**	**0.75**	**0.06218**
	NON-OBESI	**59**	**36.9**	**0.94**	**IN-SIGNIFICANT**

INTERPRETATION

Hypertensive patients with waist circumference ≥ 102 cm for males and ≥ 88 cm for females were considered obese. Obese patients have significantly high TC, TG and LDL values when compared with non-obese patients.

TABLE-12: PERCENTAGE OF DYSLIPIDEMIA:

WAIST-CIRCUMFERENCE

LIPID	' N '		PERCENTAGE	' P '
TC	**OBESE**	**48**	**64.58**	**< 0.0001**
	NON-OBESE	**59**	**27.11**	**SIGNIFICANT**
TG	**OBESE**	**48**	**87.50**	**0.3877**
	NON-OBESE	**59**	**81.35**	**IN-SIGNIFICANT**
LDL	**OBESE**	**48**	**39.58**	**0.0164**
	NON-OBESE	**59**	**18.64**	**SIGNIFICANT**
HDL	**OBESE**	**48**	**45.83**	**0.1642**
	NON-OBESE	**59**	**59.32**	**IN-SIGNIFICANT**

INTERPRETATION

Hypertensive patients who are obese with respect to waist circumference have significantly higher percentage of patients with their TC and LDL in dyslipidemic range.

TABLE-13: MEAN LIPID VALUES: WAIST-HIP RATIO (WHR)

LIPID	' N '		MEAN	SE	' P '
TC	**OBESE**	**80**	**198**	**2.97**	**0.28**
	NON-OBESE	**27**	**192**	**5.85**	**IN-SIGNIFICANT**
TG	**OBESE**	**80**	**204**	**8.93**	**0.119**
	NON-OBESE	**27**	**178**	**7.95**	**IN-SIGNIFICANT**
LDL	**OBESE**	**80**	**119**	**2.78**	**0.90**
	NON-OBESE	**27**	**120**	**5.27**	**IN-SIGNIFICANT**
HDL	**OBESE**	**80**	**38.3**	**0.70**	**0.29**
	NON-OBESE	**27**	**36.8**	**1.36**	**IN-SIGNIFICANT**

INTERPRETATION

Obesity when defined as waist hip ratio ≥ 0.85 in females and ≥ 0.90 in males did not show any significant alterations in mean lipid values when compared with non-obese hypertensive patients.

TABLE-14: PERCENTAGE OF DYSLIPIDEMIA: WAIST-HIP RATIO

LIPID	' N '		PERCENTAGE	' P '
TC	**OBESE**	**80**	**47.50**	**0.1996**
	NON-OBESE	**27**	**33.33**	**IN-SIGNIFICANT**
TG	**OBESE**	**80**	**87.50**	**0.0989**
	NON-OBESE	**27**	**74.07**	**IN-SIGNIFICANT**
LDL	**OBESE**	**80**	**28.75**	**0.51**
	NON-OBESE	**27**	**22.22**	**IN-SIGNIFICANT**
HDL	**OBESE**	**80**	**51.25**	**0.4713**
	NON-OBESE	**27**	**59.25**	**IN-SIGNIFICANT**

INTERPRETATION

Hypertensive patients with waist-hip ratio in obese range did not show any significant high percentage of dyslipidemia.

TABLE-15: MEAN LIPID VALUES:

SEDENTARY VS NON-SEDENTARY

LIPID	' N '		MEAN	SE	' P '
TC	**SEDENTARY**	**58**	**208**	**3.83**	**< 0.0001**
	NON-SEDENT	**49**	**183**	**2.59**	**SIGNIFICANT**
TG	**SEDENTARY**	**58**	**212**	**11.75**	**0.022**
	NON-SEDENT	**49**	**180**	**5.74**	**SIGNIFICANT**
LDL	**SEDENTARY**	**58**	**125**	**4.07**	**0.00924**
	NON-SEDENT	**49**	**112**	**1.98**	**SIGNIFICANT**
HDL	**SEDENTARY**	**58**	**40.1**	**0.79**	**< 0.0001**
	NON-SEDENT	**49**	**35.3**	**0.87**	**SIGNIFICANT**

INTERPRETATION

Hypertensive sedentary patients have significantly higher values of all lipids when compared with hypertensive non-sedentary patients

TABLE-16: PERCENTAGE OF DYSLIPIDEMIA:

SEDENTARY VS NON-SEDENTARY

LIPID	' N '		PERCENTAGE	' P '
TC	**SEDENTARY**	**58**	**62.06**	**< 0.0001**
	NON-SEDENT	**49**	**22.44**	**SIGNIFICANT**
TG	**SEDENTARY**	**58**	**87.93**	**0.3629**
	NON-SEDENT	**49**	**81.63**	**IN-SIGNIFICANT**
LDL	**SEDENTARY**	**58**	**37.93**	**0.0131**
	NON-SEDENT	**49**	**16.32**	**SIGNIFICANT**
HDL	**SEDENTARY**	**58**	**32.75**	**< 0.0001**
	NON-SEDENT	**49**	**77.55**	**SIGNIFICANT**

INTERPRETATION

Hypertensive sedentary patients have significantly higher percentage of individuals with TC and LDL, and significantly low percentage of individuals with HDL in dyslipidemic range.

TABLE-17: MEAN LIPID VALUES:

SOCIOECONOMIC STATUS(SES)

LIPID	' N '		MEAN	SE	' P '
TC	**SES 2 & 3**	**52**	**206**	**3.79**	**0.00052**
	SES 4 & 5	**55**	**188**	**3.36**	**SIGNIFICANT**
TG	**SES 2 & 3**	**52**	**206**	**11.85**	**0.22**
	SES 4 & 5	**55**	**189**	**7.79**	**IN-SIGNIFICANT**
LDL	**SES 2 & 3**	**52**	**125**	**4.00**	**0.0186**
	SES 4 & 5	**55**	**114**	**2.74**	**SIGNIFICANT**
HDL	**SES 2 & 3**	**52**	**39.5**	**0.86**	**0.0132**
	SES 4 & 5	**55**	**36.4**	**0.86**	**SIGNIFICANT**

INTERPRETATION

Hypertensive patients of SES 2&3 have significantly high mean TC, LDL and HDL values when compared with hypertensive patients of SES 4&5.

TABLE-18: PERCENTAGE OF DYSLIPIDEMIA:

SOCIO-ECONOMIC STATUS

LIPID	' N '		PERCENTAGE	' P '
TC	**SES 2 & 3**	**52**	**61.53**	**0.0004**
	SES 4 & 5	**55**	**27.27**	**SIGNIFICANT**
TG	**SES 2 & 3**	**52**	**84.61**	**0.8963**
	SES 4 & 5	**55**	**83.63**	**IN-SIGNIFICANT**
LDL	**SES 2 & 3**	**52**	**40.38**	**0.0057**
	SES 4 & 5	**55**	**16.36**	**SIGNIFICANT**
HDL	**SES 2 & 3**	**52**	**42.3**	**0.027**
	SES 4 & 5	**55**	**63.63**	**SIGNIFICANT**

INTERPRETATION

Hypertensive patients of SES 2&3 have significantly high percentage of individuals with TC and LDL in dyslipidemic range and significantly low percentage individuals with HDL in dyslipidemic range.

TABLE-19: MEAN LIPID VALUES: STAGES OF HYPERTENSION

LIPID	' N '		MEAN	SE	' P '
TC	**STAGE - 1**	**32**	**191**	**4.02**	**0.19**
	STAGE - 2	**75**	**199**	**3.37**	**IN-SIGNIFICANT**
TG	**STAGE - 1**	**32**	**195**	**15.73**	**0.86**
	STAGE - 2	**75**	**198**	**7.54**	**IN-SIGNIFICANT**
LDL	**STAGE - 1**	**32**	**114**	**4.22**	**0.153**
	STAGE - 2	**75**	**121**	**2.98**	**IN-SIGNIFICANT**
HDL	**STAGE - 1**	**32**	**37.8**	**1.13**	**0.899**
	STAGE - 2	**75**	**38.0**	**0.76**	**IN-SIGNIFICANT**

INTERPRETATION

There is no significant difference in mean lipid values between patients in stage-1 and stage-2 hypertension.

TABLE-20: PERCENTAGE OF DYSLIPIDEMIA:

STAGES OF HYPERTENSION

LIPID	' N '		PERCENTAGE	' P '
TC	**STAGE -1**	**32**	**43.75**	**1.00**
	STAGE - 2	**75**	**44.00**	**IN-SIGNIFICANT**
TG	**STAGE -1**	**32**	**84.37**	**0.9748**
	STAGE - 2	**75**	**84.00**	**IN-SIGNIFICANT**
LDL	**STAGE -1**	**32**	**28.12**	**1.00**
	STAGE - 2	**75**	**28.00**	**IN-SIGNIFICANT**
HDL	**STAGE -1**	**32**	**50.00**	**0.6596**
	STAGE - 2	**75**	**54.66**	**IN-SIGNIFICANT**

INTERPRETATION

There is no significant difference in percentage prevalence of dyslipidemia among stage-1 and stage-2 hypertensives.

TABLE-21:MEAN LIPID VALUES

PRE-DIABETIC VS NON DIABETIC

LIPID	' N '		MEAN	SE	' P '
TC	**PRE-DIABET**	**35**	**206**	**4.61**	**0.0144**
	NON-DIABET	**72**	**192**	**3.15**	**SIGNIFICANT**
TG	**PRE-DIABET**	**35**	**208**	**14.56**	**0.31408**
	NON-DIABET	**72**	**192**	**7.70**	**IN-SIGNIFICAN**
LDL	**PRE-DIABET**	**35**	**125**	**4.83**	**0.074**
	NON-DIABET	**72**	**116**	**2.74**	**IN-SIGNIFICAN**
HDL	**PRE-DIABET**	**35**	**38.8**	**1.15**	**0.3543**
	NON-DIABET	**72**	**37.5**	**0.76**	**IN-SIGNIFICAN**

INTERPRETATION

Hypertensive pre-diabetic patients have significantly high mean total cholesterol when compared with hypertensive non-diabetic patients.

TABLE-22: PERCENTAGE OF DYSLIPIDEMIA:

PRE-DIABETIC VS NON-DIABETIC

LIPID	' N '		PERCENTAGE	' P '
TC	**PRE-DIABET**	**35**	**62.85**	**0.0059**
	NON-DIABET	**72**	**34.72**	**SIGNIFICANT**
TG	**PRE-DIABET**	**35**	**82.85**	**0.8081**
	NON-DIABET	**72**	**84.72**	**IN-SIGNIFICANT**
LDL	**PRE-DIABET**	**35**	**42.85**	**0.0173**
	NON-DIABET	**72**	**20.83**	**SIGNIFICANT**
HDL	**PRE-DIABET**	**35**	**42.85**	**0.1323**
	NON-DIABET	**72**	**58.33**	**IN-SIGNIFICANT**

INTERPRETATION

Significantly higher percentages of pre-diabetic hypertensive patients have their total cholesterol and LDL cholesterol in dyslipidemic range when compared with non-diabetic hypertensive patients.

DISCUSSION

PREVALENCE OF DYSLIPIDEMIA

On analysis of the lipid profile of 107 hypertensive patients and 60 normotensive persons the mean TC values in cases and controls are 197 mg/dl and 166 mg/dl respectively. The mean TG values are 197 mg/dl and 120 mg/dl, the mean LDL c values are 119 mg/dl and 98.4 mg/dl . All these differences are statistically significant with a 'p' value of < 0.0001 when analyzed with unpaired T test.The mean HDL (37.9 mg/dl) in hypertensive is significantly lower (p <0.0001) than normotensive (42.3 mg/dl).

About 43.92 % of hypertensive has high TC (i.e. ≥200 mg/dl) when compared with the normotensives (i.e. 5%). High TG (≥150 mg/dl) is found in 84.11 % of the hypertensive population , whereas it is seen only in 20% of normotensives . The high LDL in the groups is 28.03% and 5%. The low HDL (<40 mg/dl) is seen in 53.27 %of hypertensive and 30% of normotensive. All these values are statistically significant when analyzed using Chi-square test.

The results are similar to the studies conducted in Nigeria by J.Idemudia E.Ugwuja [63] which showed a significantly elevated plasma TC, TG, LDL-c and HDL-c in hypertensive patients when compared with

normotensive patients. Studies conducted by M.S . Saha, N .K. Sana and Ranajith kumar Shaha [64] in northern Bangladesh also showed a significantly high TC , TG and LDL values (TC-291.25 mg/dl vs. 182.14 mg/dl, TG-184.77 mg/dl vs. 142.73 mg/dl and LDL-154.32 mg/dl vs. 105.73 mg/dl) and significantly lower HDL-c values (32.91 mg/dl vs . 42.88 mg/dl) in hypertensive patients when compared with normotensive patients. Studies by Abdishakur Abdulla, Nico Negelkerke [65] in UAE showed a significantly higher level of VLDL and TG among hypertensive patients but not TC and LDL levels. Studies conducted in Spain by D.Rueda and A.Maldonado [66] showed a significantly high TC and TG than normotensive controls.

INFLUENCE OF AGE

The hypertensive patients included in our study were divided into three age groups (31-45yrs, 46-60yrs, and 61-75yrs) and the mean lipid values of the group were compared. The TC were significantly higher in hypertensive of the group-III when compared with the group-I (mean TC 203 mg/dl vs. 189 mg/dl, p=0.049). The TG, LDL and HDL did not show any significant differences. On analyzing the percentage of dyslipidemia in each group , the group - I had significantly higher percentage of patients

with TC in dyslipidemic range (53.19% vs. 20%, p=0.0151) when compared with group-III

INFLUENCE OF SEX

In our study hypertensive males have significantly lower mean HDL levels when compared with hypertensive females (HDL 36.4 mg/dl vs . 39.7 mg/dl, p=0.0084). About 63.79% of males were in the dyslipidemic HDL range , when compared with females (40.08%) , the value is significant (p=0.0084) . Other cholesterols were higher in males but not significantly so.

Study conducted by S.A.Desai, U . V .Mani , S. M . Deshmukh, U.M .Iyer, A.K.Sen and R.P.Patel[67] in Gujarat showed hypertensive males have significantly higher TC (200 mg/dl vs. 175.5 mg/dl) , TG (176.5 mg/dl vs. 157.3 mg/dl) and LDL (128.1mg/dl vs. 107.7 mg/dl) levels. This favorable profile in females was probably due to the influence of estrogen hormone. In contrast to our study this study showed a significantly higher HDL values in hypertensive males (40.7 mg/dl vs. 32.5 mg/dl).

In Nigerian study by J.Idemudia and E.Ugwuja, the TC was significantly higher in hypertensive females (4.45 mmol/L vs . 4.86

mmol/L, p=<0.05) than hypertensive males. The other lipids including HDL-c were higher in females but not in the significant range.

The study of northern Bangladesh showed higher TC, TG, LDL-c and low HDL-c levels in hypertensive males but the values were not significant.

INFLUENCE OF SMOKING

The mean TC, TG, LDL and HDL values in our study were higher in hypertensive smokers when compared with hypertensive non-smoker males. (Mean values: TC-215 mg/dl vs. 188 mg/dl, TG-233 mg/dl vs. 182 mg/dl, LDL-129 mg/dl vs. 117 mg/dl and HDL- 39.3 mg/dl vs. 34.7 mg/dl). Among these except LDL all values were statistically significant. The percentage of dyslipidemia is higher among the smoker population with respect to all lipid parameters, but only the TC was significant.

In the study conducted by Jaroslaw Goldman and Marian Klinger[68] of Poland the hypertensive smokers showed higher mean TC (6.23 mmol/L vs. 5.57 mmol/L), LDL-c (3.80 mmol/L vs . 3.76 mmol/L) , TG (2.53 mmol/L vs . 1.60 mmol/L) and HDL-c (1.18 mmol/L vs. 1.13 mmol/L). Among these TC and TG were statistically significant . The

results were exactly similar to our study. The study by Mojgan Gharipour and Roya Kelishadi [69] also showed a significantly higher TG and LDL-c values (p=0.005 and 0.015 respectively) and insignificant but high values of TC (p=0.079) and insignificant but low value of HDL. A study by S. A .Desai and U. V .Mani in Baroda showed a significantly high TG and a insignificantly high TC values among smokers. Other lipids did not show much difference.

The proposed mechanisms by which smoking alters the lipid profile are [70]

- Nicotine stimulates the release of adrenaline, leading to increased serum concentrations of FFA.
- FFA is a stimulant of hepatic secretion of VLDL and hence TG.
- HDL-c varies inversely with VLDL-c in serum.
- FFA also stimulates hepatic synthesis and secretion of cholesterol.
- Smoking induces cytochrome p-450 system that degrades anti HT drugs[71].

INFLUENCE OF OBESITY

Body Mass Index (BMI)

In our study obese patients when defined with BMI of ≥ 25 kg/m² showed significantly high values of TC, TG, LDL and HDL (p values

TC- < 0.0001, TG- 0.011 , LDL- 0.0035 and HDL- < 0.0001). The percentage of dyslipidemia is also significantly higher among obese patients with respect to TC, LDL and HDL and insignificantly high with respect to TG.

Study by Muhammad. S. Akthar and Sayeda.M.Ansar in Faisalabad[72] pakistan showed TC was significantly high among obese hypertensives and LDL, TG and HDL were high but not significant . The study by S .A .Desai and U.V.Mani in Baroda also showed the significantly high lipid profile of TC , TG , LDL and HDL among obese hypertensives.The high HDL among obese patients may be due to ample fruit intake and vegetable intake.

Waist Circumference

Patients with obesity when defined as waist circumference of ≥ 88cm in females and ≥102cm in males showed significantly high values of TC (p=0.00015), TG (p=0.024), LDL(p=0.019) and high but insignificant (p=0.063) value of HDL.

The percentage of dyslipidemia was significantly high with respect to TC and LDL and insignificantly high with respect to TG and insignificantly low with respect to HDL.

Waist hip ratio (WHR)

Patients with obesity when defined by WHR ≥ 0.85 in females and ≥ 0.9 in males showed high TC, TG and LDL values and low HDL values, but none of them were significant. The percentage of dyslipidemia p revalence also showed similar trend.

INFLUENCE OF PHYSICAL ACTIVITY.

In our study hypertensive patients who were sedentary had significantly high TC, TG and LDL and significantly low HDL values.Study by Ignez Salas Martius and Teixeira Coelho in Brazil also showed similar pattern of lipid abnormalities.

INFLUENCE OF SOCIO-ECONOMIC STATUS.

The mean TC, TG and LDL are high and mean HDL values are low among SES class 2&3 hypertensive patients when compared with hypertensive patients of SES class 4&5 (p values TC- 0.00052, TG- 0.22, LDL- 0.0186 and HDL- 0.0132), all these values are significant except for TG. The percentage of prevalence also showed similar pattern. The high prevalence of dyslipidemia in h igh socio-economic group is probably due to their sedentary life style and increased intake of fatty foods.

INFLUENCE OF STAGES OF HYPERTENSION.

Comparison of lipid profile of stage - I hypertensive patients with stage - Π hypertensive patients did not show any significant difference in mean values and percentage prevalence. A study conducted by S.Sharif, A.Cheema and M.Khan[73] at Lahore showed significantly high values of TC and LDL among stage-Π hypertensive but no significant difference in mean values of HDL and TG.

INFLUENCE OF PRE-DIABETIC STATE.

The lipid profile of hypertensive patients with their blood sugar values in pre – diabetic range showed high mean values of TC, TG, LDL and HDL when compared with the values of hypertensive patients with normal blood sugar. Among these only TC was significantly high.

CONCLUSIONS

1. Hypertensive patients have significantly higher levels of all forms of cholesterol and higher percentage of individuals in dyslipidemic state when compared with normotensive persons.

2. Hypertensive females have significantly higher levels of HDL c when compared to hypertensive males.

3. Elderly hypertensives have significantly high total cholesterol values when compared with young and middle aged hypertensives.

4. Smoking have a significant impact on the lipid profile of hypertensives.

5. Obesity when calculated using Body Mass Index and Waist Circumference correlates positively with abnormal lipid profile in hypertensives, whereas the Waist Hip Ratio does not show any correlation.

6. Hypertensives who are sedentary and and in high Socio Economic Status have high prevalence of dyslipidemia.

7. The stage of hypertension does not alter the lipid profile in hypertensives.

8. Pre-Diabetic state significantly increases the total cholesterol in hypertensive patients.

LIMITATIONS

1. The sample size is small.

2. The design of the study is cross sectional.

3. The impact of treatment of dyslipidemia on hypertension and vice versa could not be studied longitudinally.

4. Since most of the patients who attend the hospital OP department belong to low socio economic group, the pattern of dyslipidemia in high socio economic group could not be studied.

5. Chances of confounding biases are more.

6. The emerging risk factors like Lipoprotein (a) and LDL c sub-fractions are not included in the study.

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ABBREVIATIONS

ATP II	- Adult treatment and panel III
BP	- Blood Pressure
BUN	- Blood Urea Nitrogen
BMI	- Body mass Index
CAD	- Coronary Artery Disease
CHD	- Coronary Heart Disease
CVA	- Cerebrovascular Accident
ECG	- Electrocardiography
ESRD	- End Stage Renal Disease
ET	- Endothelin
FBS	- Fasting blood sugar
FFA	- Free Fatty Acid
HDL-c	- High Density lipoprotein Cholesterol
IHD	- Ischemic Heart Disease.
LDL-c	- Low Density Lipoprotein Cholesterol
MRFIT	- Multiple risk factor intervention trial.
NO	- Nitric Oxide.
SBP	- Systolic Blood pressure.
SES	- Socio Economic Status
TC	- Total Cholesterol
TG	- Triglycerides
TIA	- Transient Ischemic Attack
VLDL	- Very Low Density Lipoprotein
WHR	- Waist Hip Ratio

LIPID PROFILE IN HYPERTENSIVE PATIENTS

PROFOMA

Case	
Control	

No: HT OP No:

Name: Age: Sex:

Address: Phone No:

Socio-Economic status : (by modified Kuppuswamy scale)

Class	I	II	III	IV	V

Life style : (physical activity ≤ 2 MET hour / day)

Sedentary	Non Sedentary

H/O smoking : (≥ 5 cigarettes / day)

Yes	No

Family H/o Hypertension

Father	Mother	Siblings

Anthropometry

Height (in cm)	Weight (in kg)	BMI
Waist (in cm)	Hip (in cm)	WHR

Blood Pressure

Sitting	R UL	L UL
Supine		

Investigations

Blood Sugar

Fasting -

PP -

B. Urea -

S. Creatinine -

Lipid profile

TC -

HDL -

LDL -

VLDL-

TG -

Modified (2007) Kuppuswamy socioeconomic status scale

EDUCATION

Profession / Honors	7	
Graduate / PG	6	
Post high school / diploma	5	
High school certificate	4	
Mid school certificate	3	
Primary certificate	2	
Illiterate	1	

OCCUPATION

Profession	10	
Semi – profession	6	
Clerical / shop owner/ farmer	5	
Skilled worker	4	
Semi skilled worker	3	
Unskilled worker	2	
Unemployed	1	

Family income / month

≥ 19575	12	
9788- 19574	10	
7323 – 9787	6	
4894 – 7322	4	
2936 – 4893	3	
980 – 2935	2	
< 980	1	

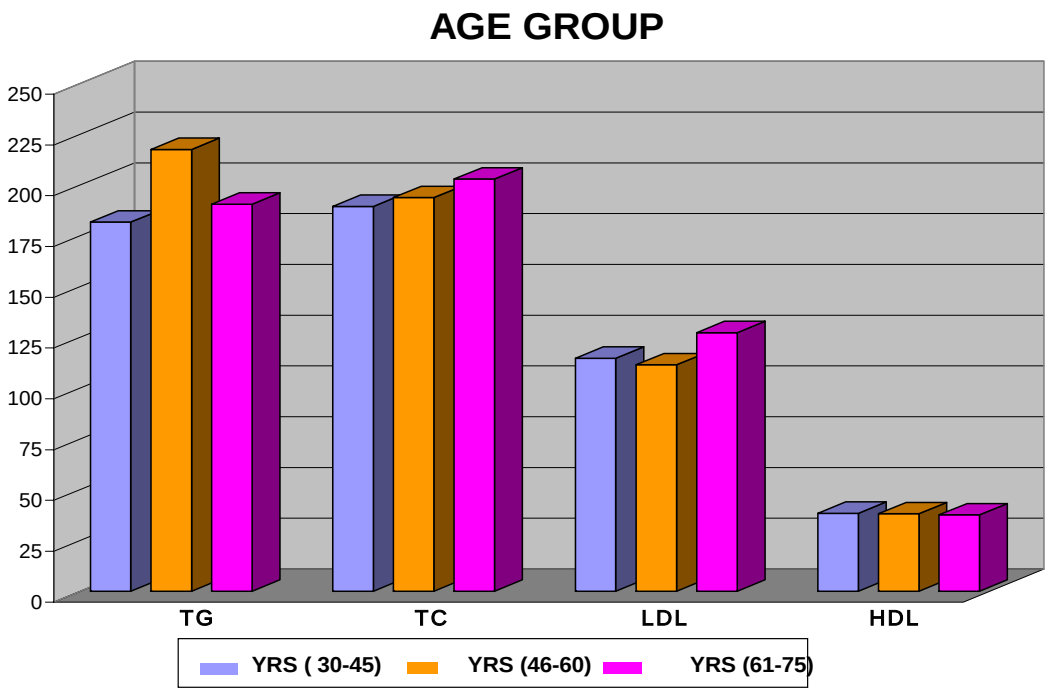
Total Points	Class
26 - 29	Upper – I
16 - 25	Upper middle - II
11 - 15	Lower middle - III
5 - 10	Upper lower – IV
< 5	Lower V
Patients	points class

Physical activity assessment

Activity	MET	Duration	MET calculation
Washing / House keeping/ driving / cooking	1-2		
Carpentry / walking (4mph) / Dancing	3-5		
Bigging in garden / Tennis games / swimming / cycling 10mph	5-7		
Tracking / jogging	7-9		
Carrying loads / walking uphill / running	>9		
Total MET			

Life Style

Sedentary	Non - sedentary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TG
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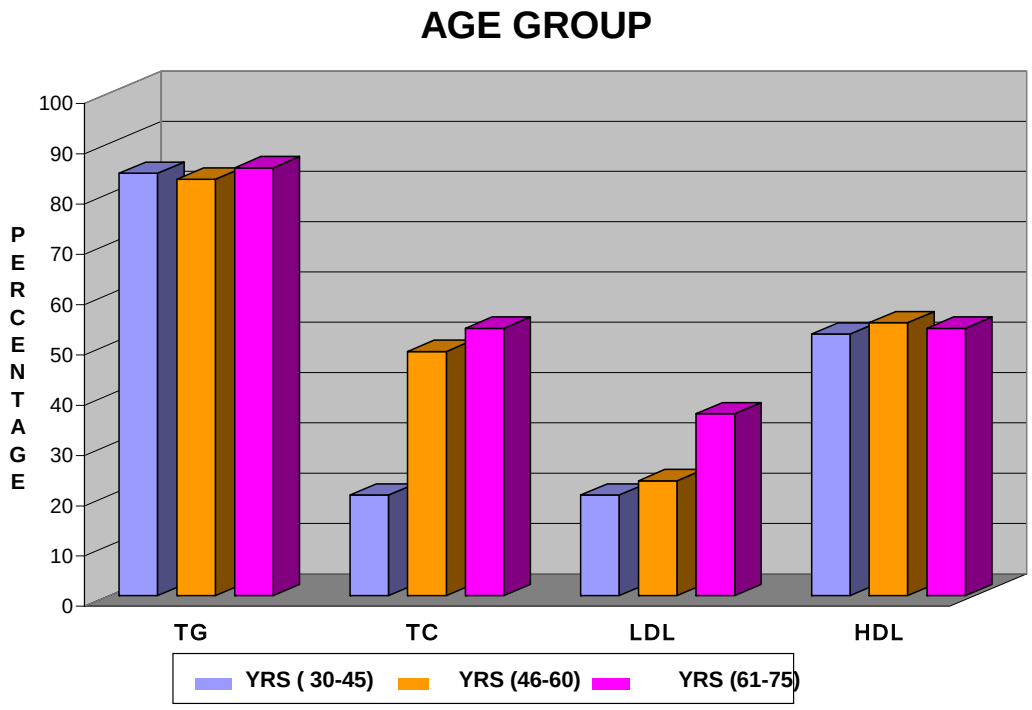
YRS (30-45)
YRS (46-60)
YRS (61-75)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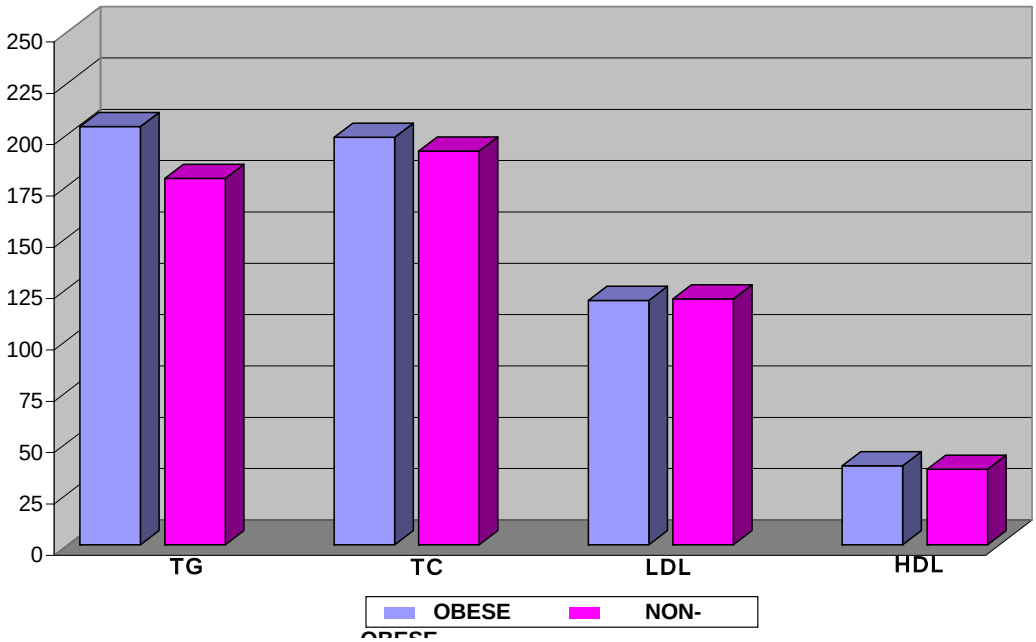
YRS (30-45)
YRS (46-60)
YRS (61-75)

WAIST-HIP RATIO



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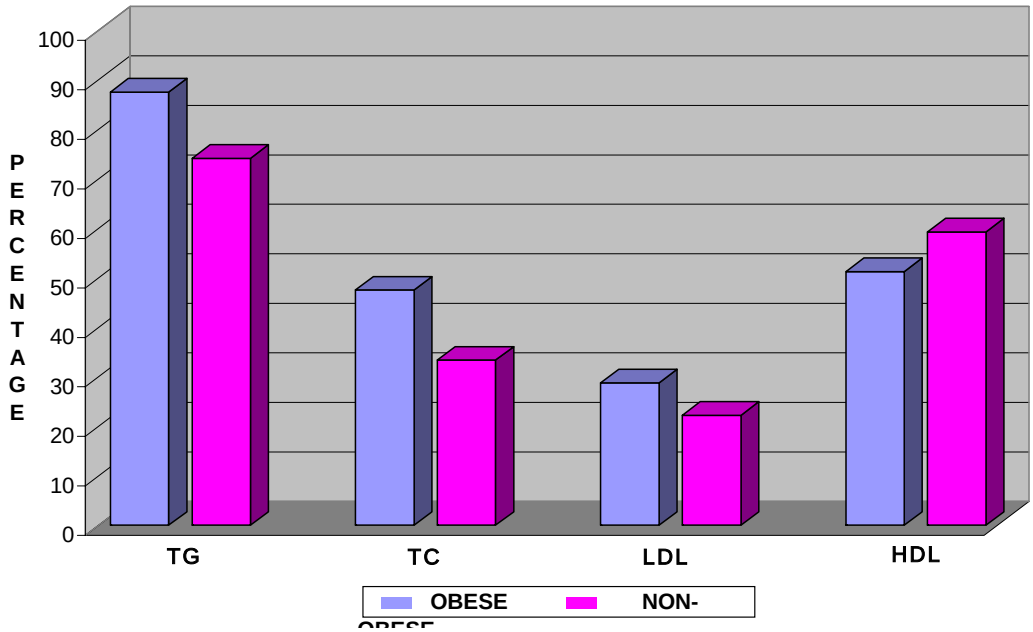

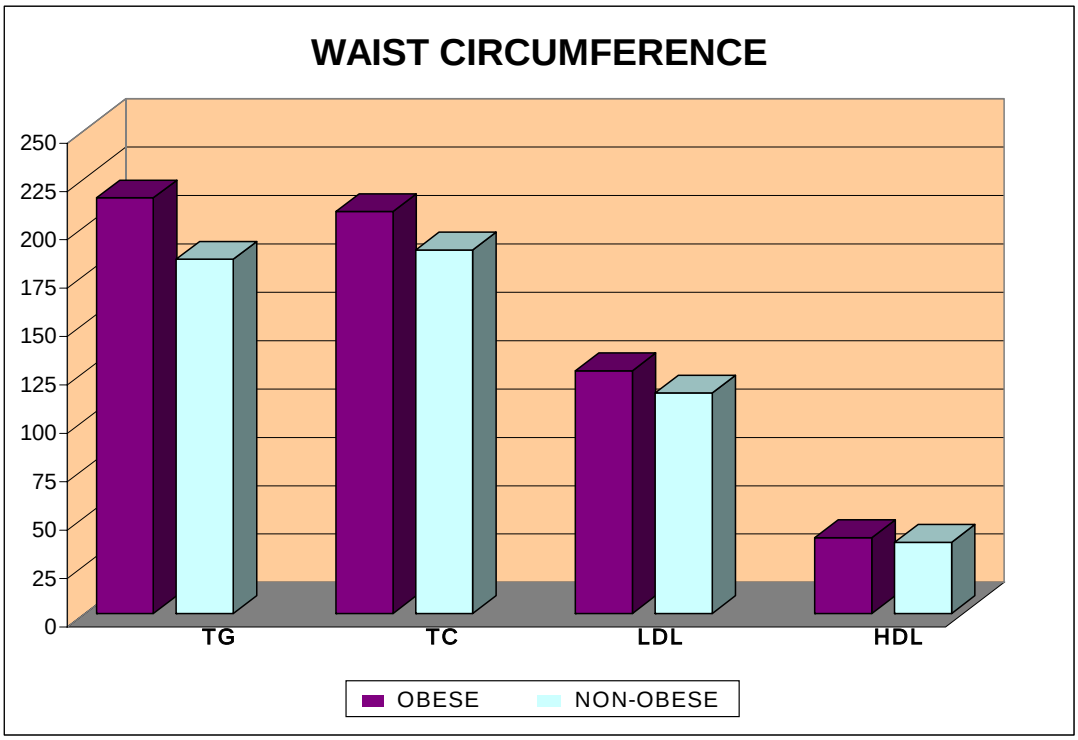
WAIST CIRCUMFERENCE
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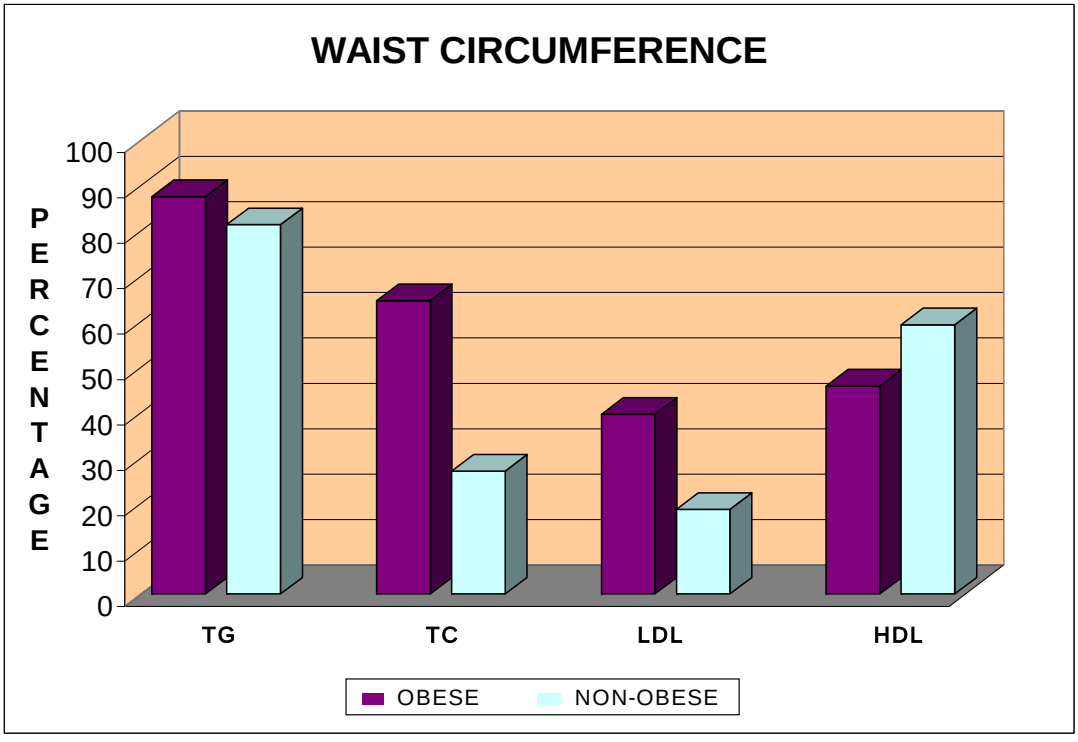
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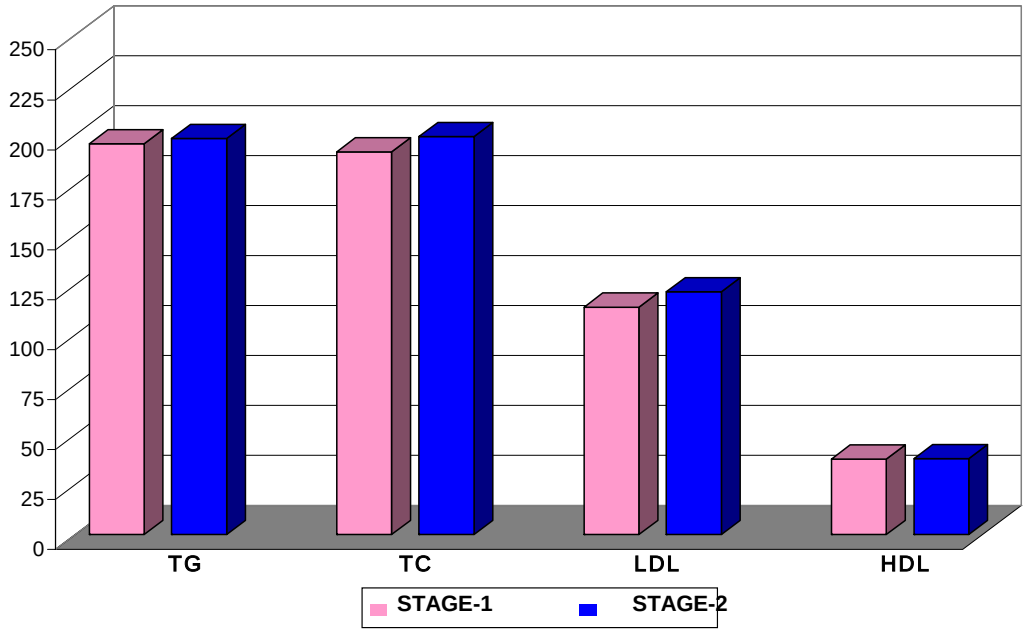
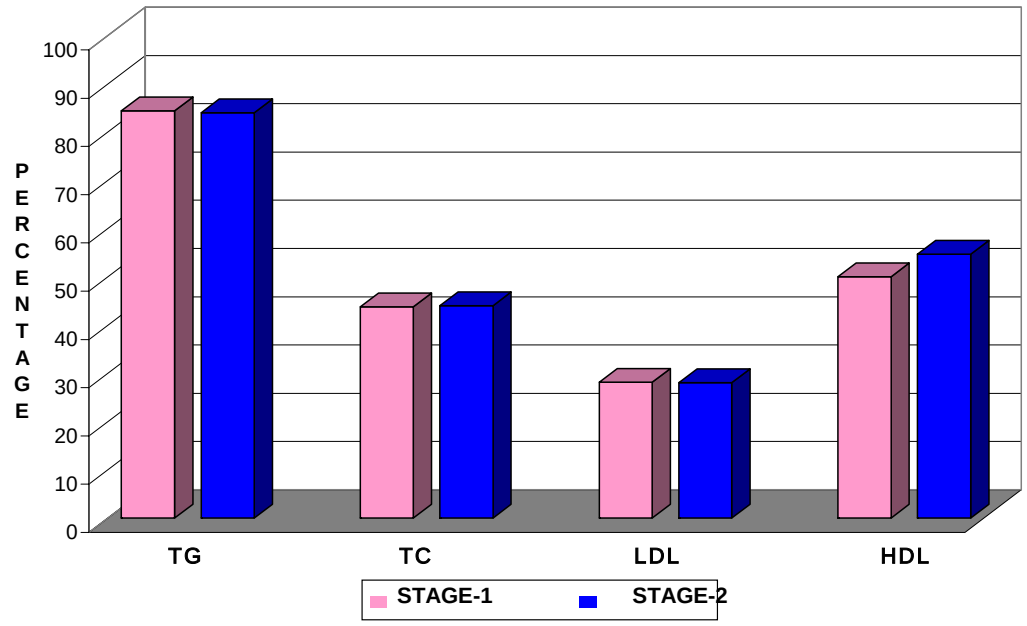
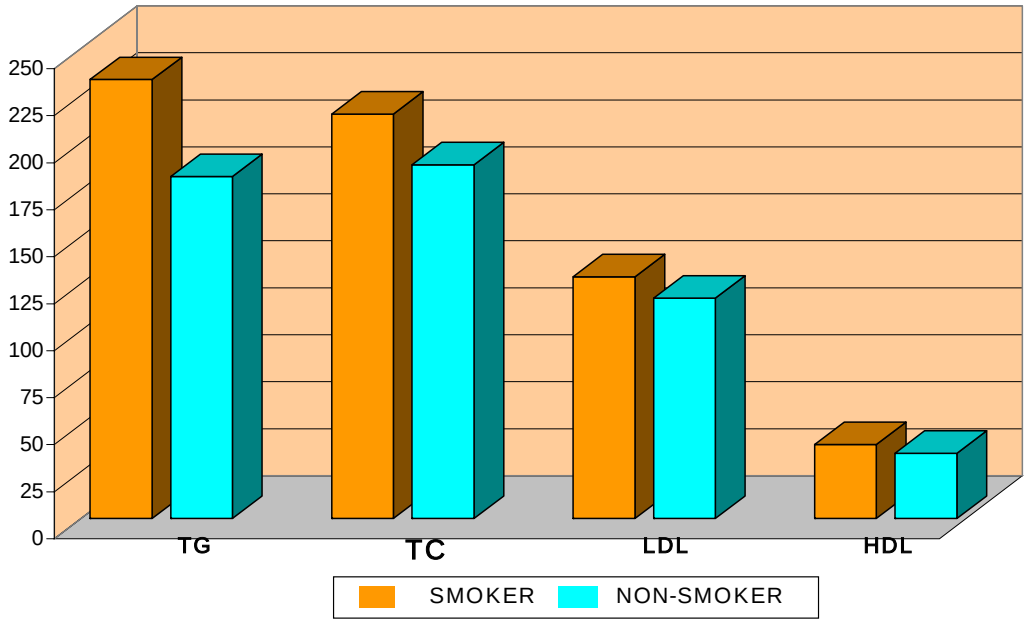
STAGES OF HT



STAGES OF HT



SMOKERS VS NON-SMOKERS



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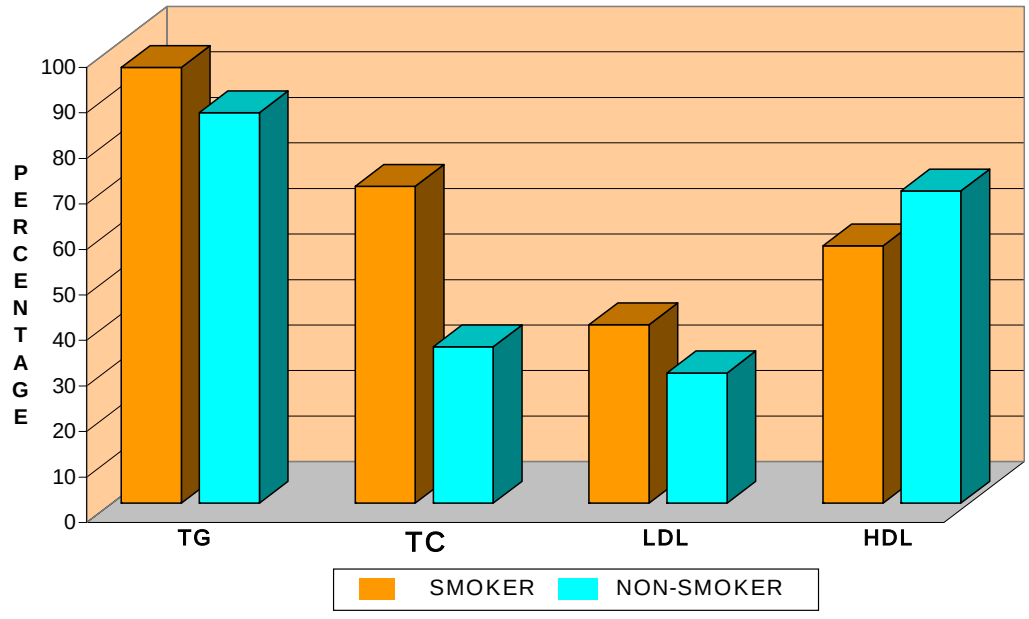

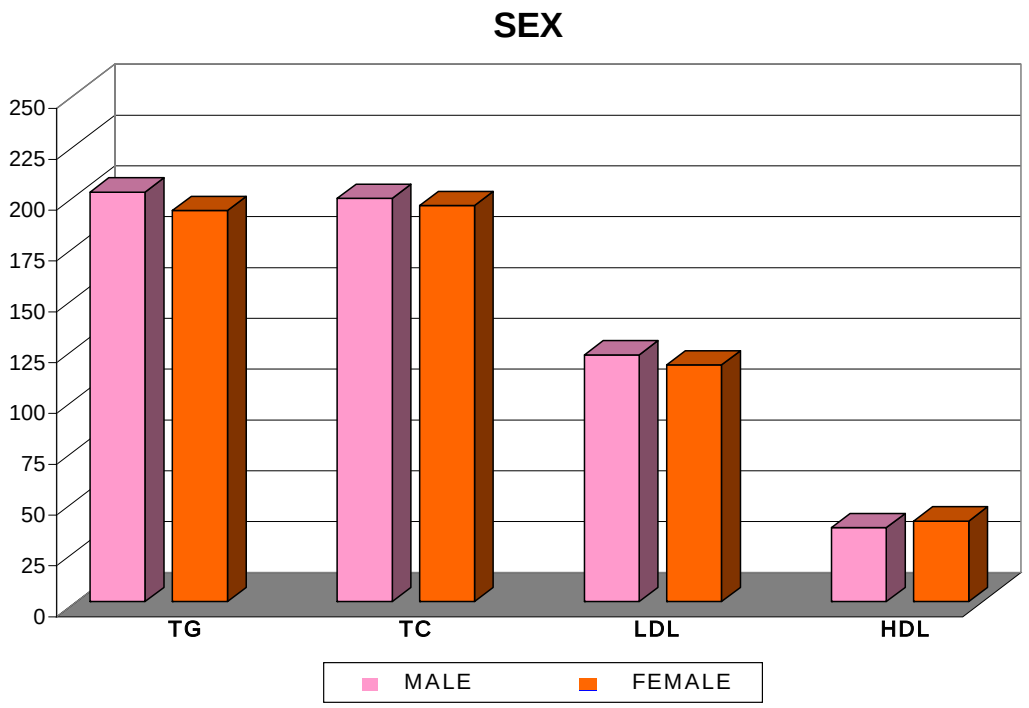
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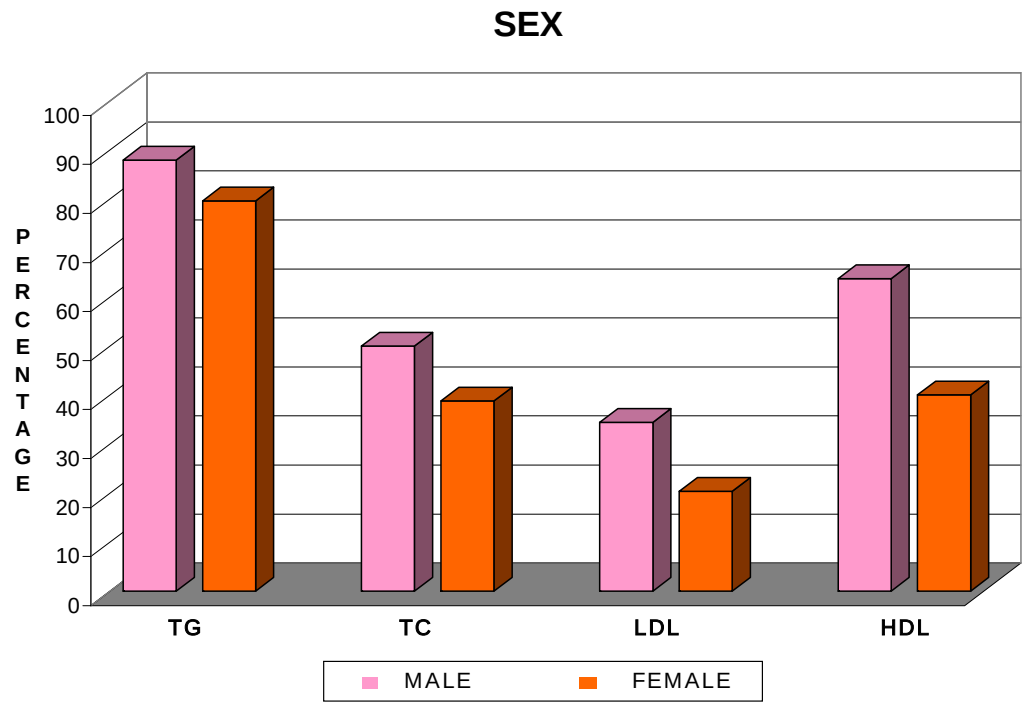
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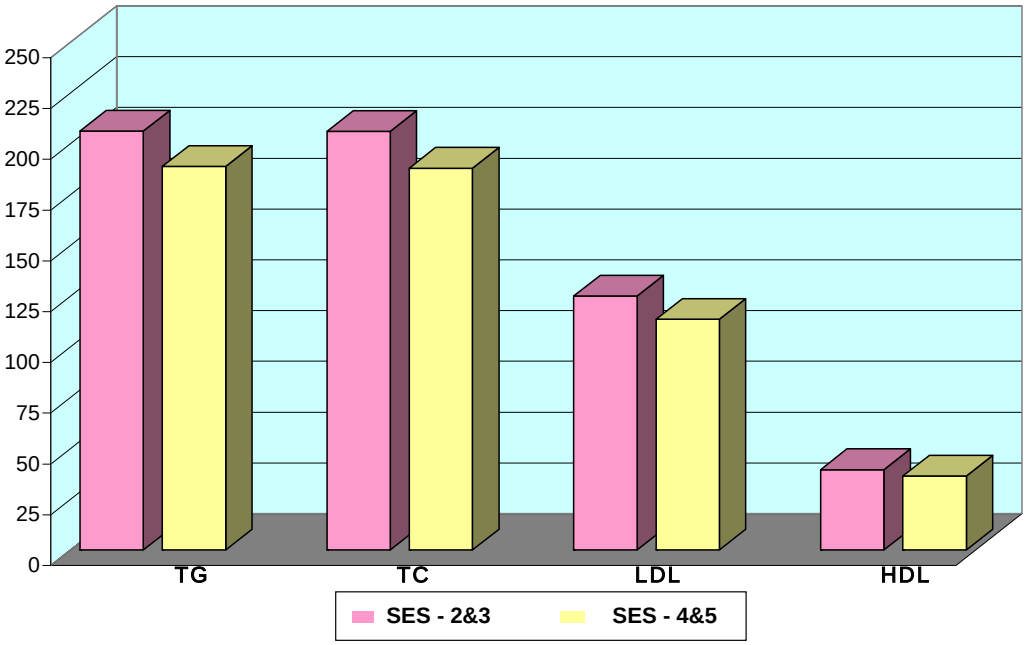
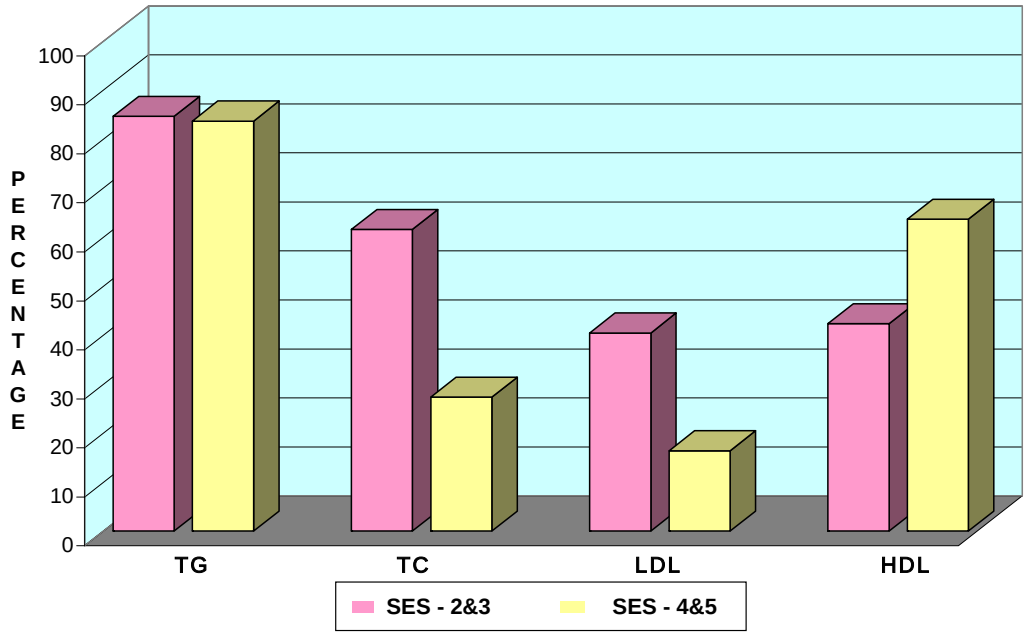
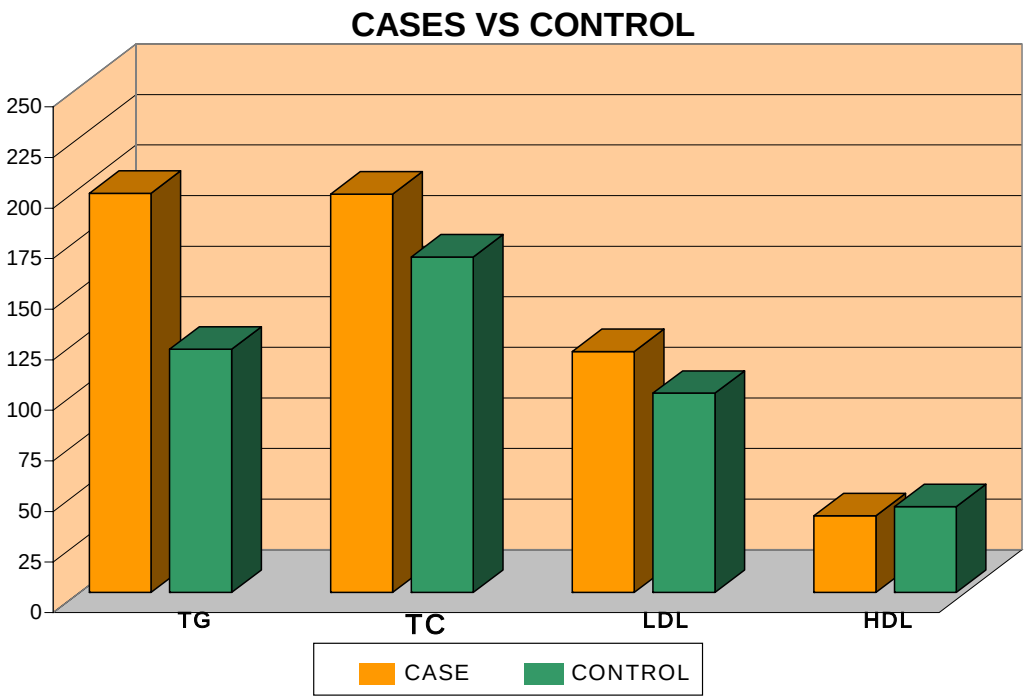
SOCIO-ECONOMIC STATUS



SOCIO-ECONOMIC STATUS

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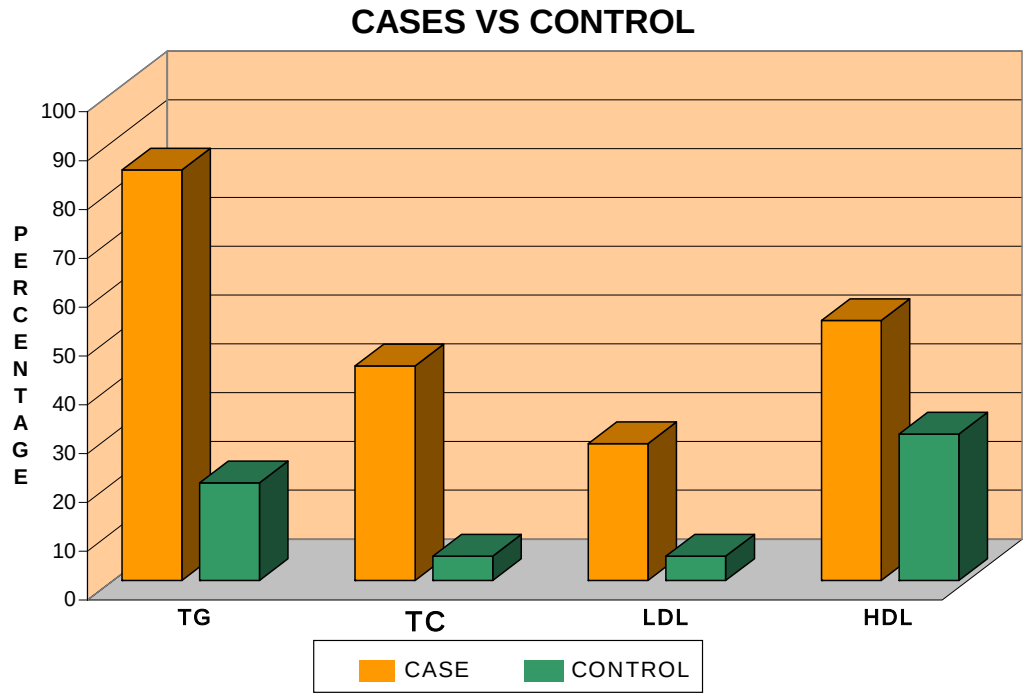
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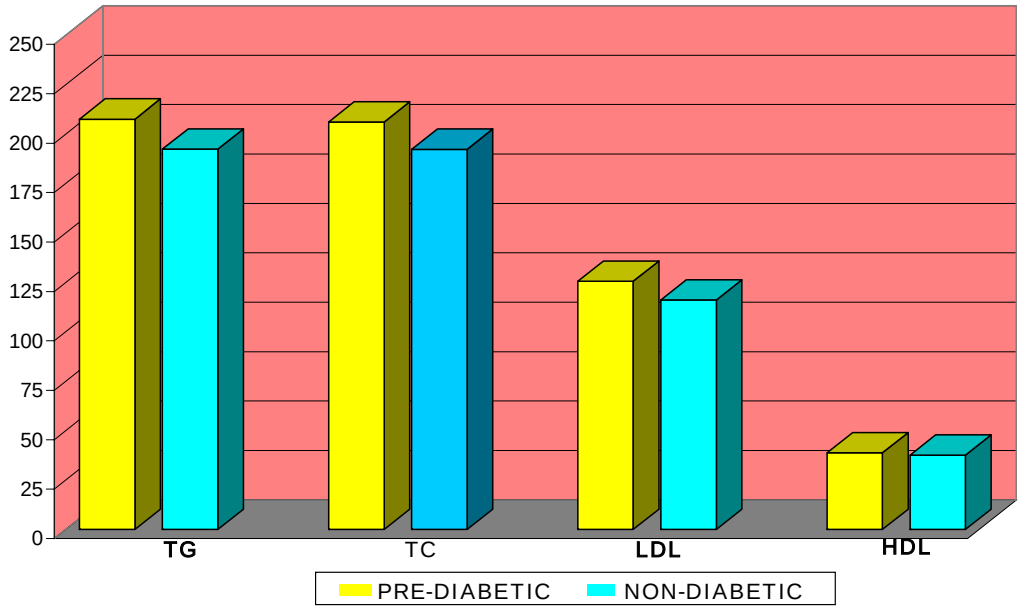
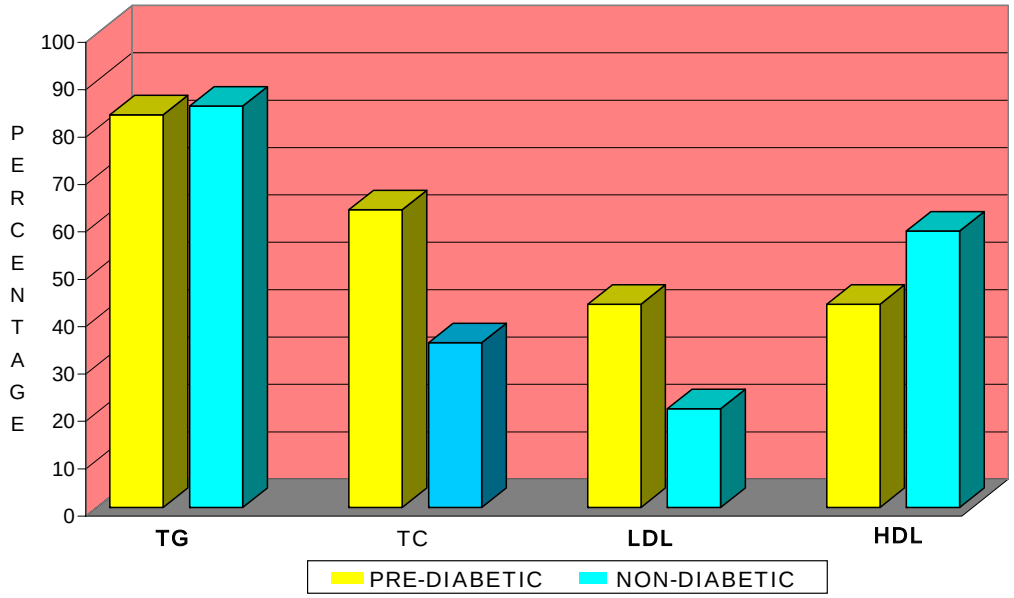
60
50
40
30
20
10
0
TG
TC
LDL
HDL
CASE
CONTROL

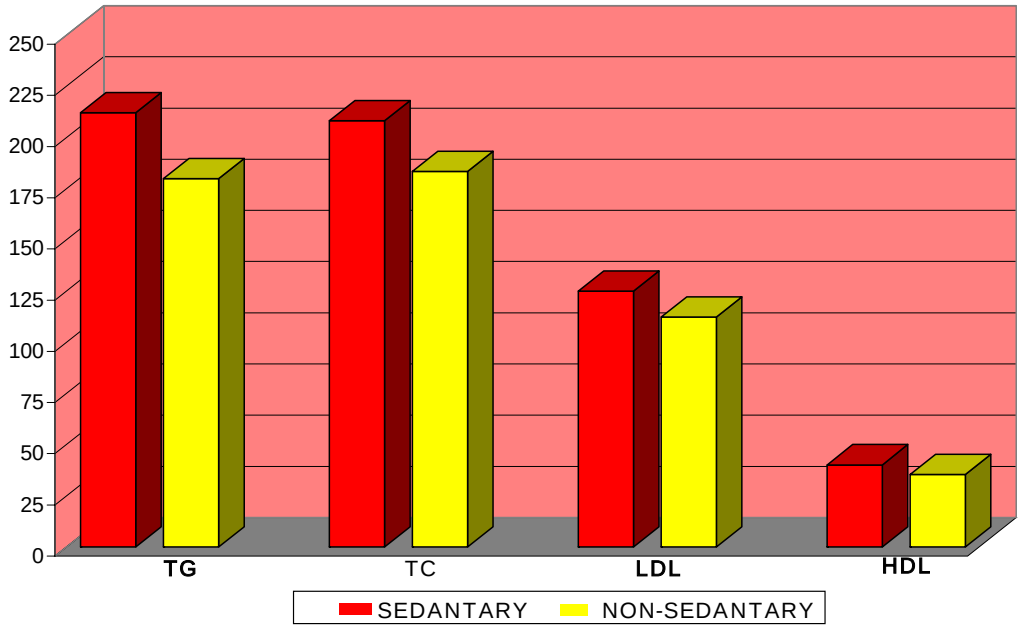
PRE-DIABETIC VS NON-DIABETIC



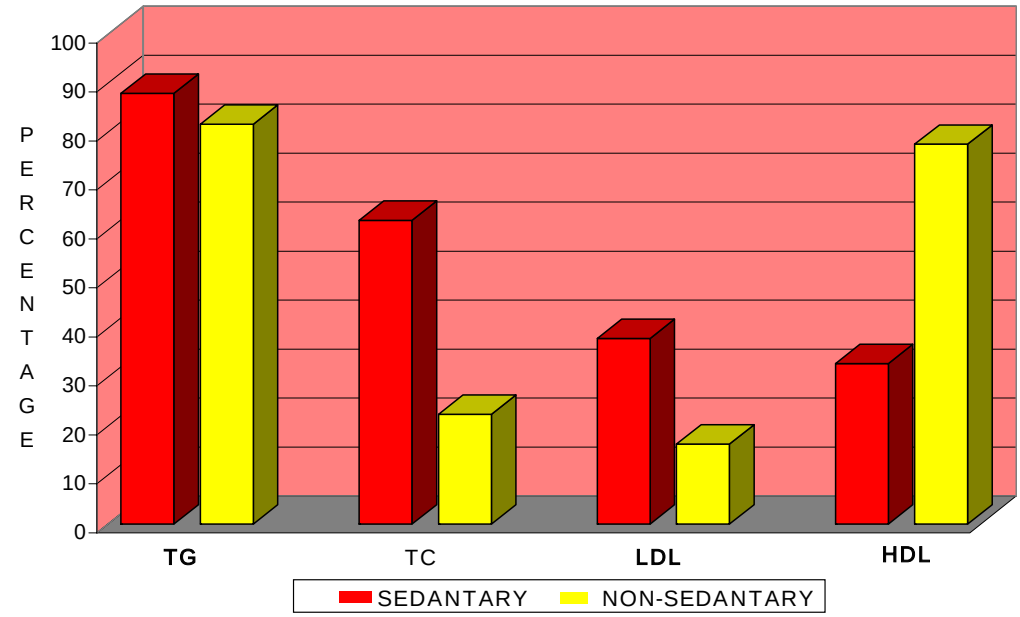
PRE-DIABETIC VS NON-DIABETIC

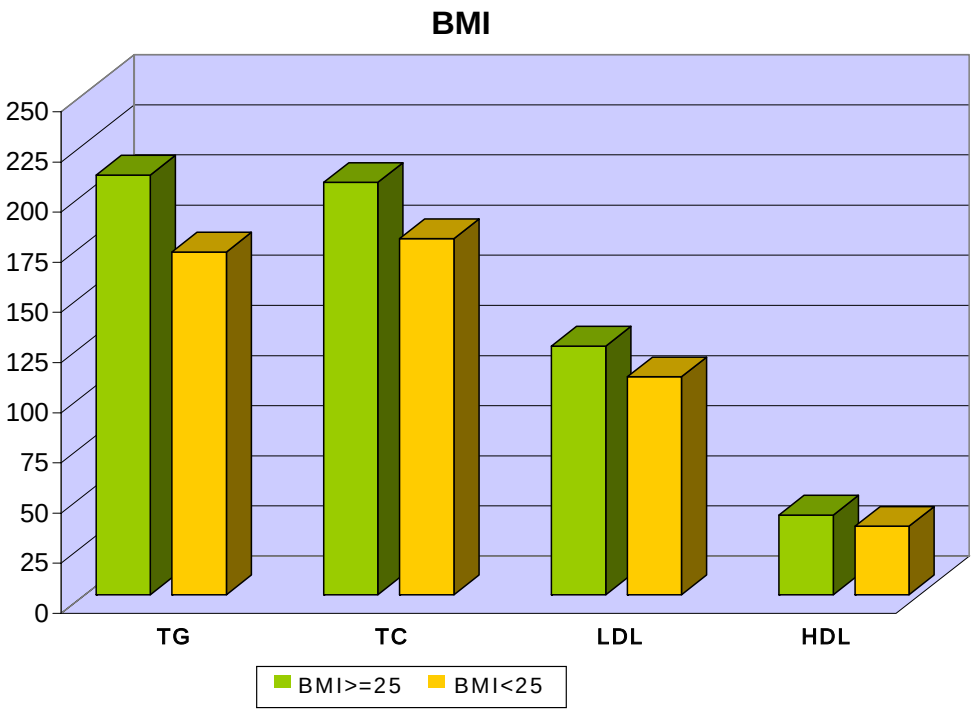


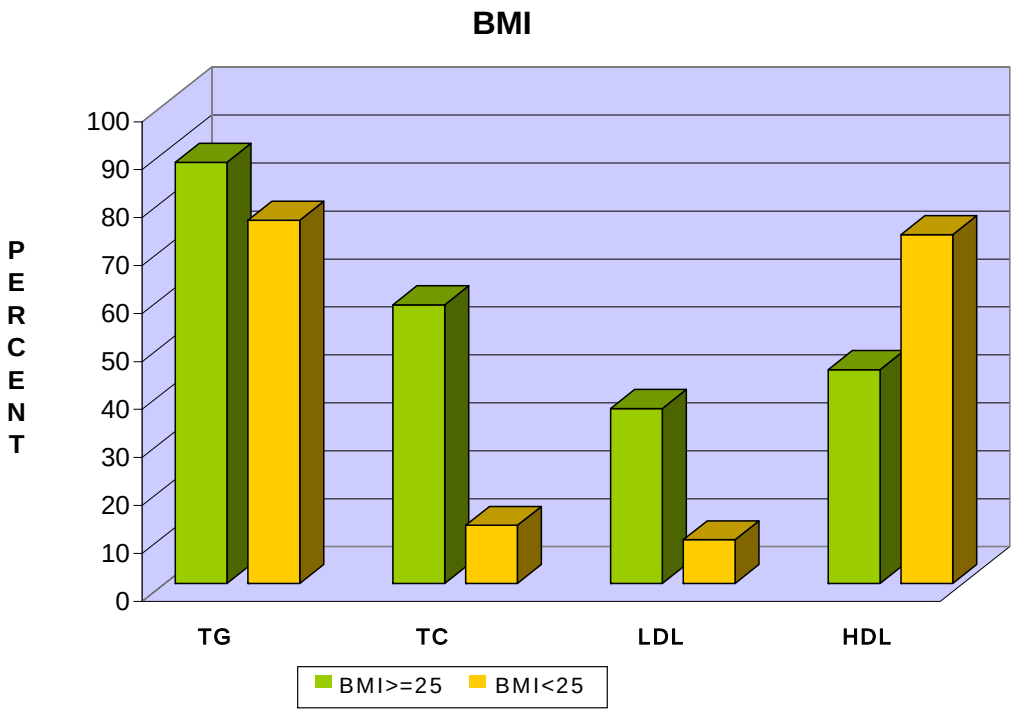
LIFE STYLE



LIFE STYLE




BMI
250
225
200
175
150
125
100
75
50
25
0
TG
TC
LDL
HDL
BMI>=25
BMI<25


BMI
PERCENT
100
90
80
70
60
50
40
30
20
10
0
TG
TC
LDL
HDL
BMI>=25
BMI<25

MASTER CHART – CASES .TOTAL NO - 107

SL.NO	AGE	SEX	SES	LS	SMOKING	HEIGHT	WEIGHT	BMI	WAIST	HIP	WHR
1	50	M	5	N	-	170	60	20.76125	91	98	0.928571
2	56	M	5	N	-	158	65	26.03749	102	100	1.02
3	51	M	5	N	-	168	56	19.84127	88	94	0.93617
4	49	M	4	N	-	172	62	20.95727	98	109	0.899083
5	57	M	5	N	-	160	66	25.78125	87	102	0.852941
6	60	M	4	S	-	170	86	29.75779	106	108	0.981481
7	57	M	5	N	-	170	61	21.10727	88	108	0.814815
8	49	M	4	N	-	160	79	30.85938	104	102	1.019608
9	51	M	5	S	-	168	80	28.34467	110	102	1.078431
10	60	M	4	N	-	171	63	21.54509	92	98	0.938776
11	68	M	3	S	-	167	76	27.25089	103	101	1.019802
12	68	M	4	N	-	170	63	21.79931	92	98	0.938776
13	67	M	3	S	-	158	68	27.23922	116	109	1.06422
14	71	M	4	N	-	164	56	20.82094	88	96	0.916667
15	62	M	3	N	-	161	74	28.54828	109	112	0.973214
16	62	M	4	N	-	174	62	20.47827	92	99	0.929293
17	63	M	3	N	-	162	63	24.00549	90	98	0.918367
18	64	M	4	N	-	170	65	22.49135	86	96	0.895833
19	62	M	3	N	-	154	64	26.986	92	98	0.938776
20	67	M	5	S	-	158	65	26.03749	106	104	1.019231
21	75	M	3	N	-	164	58	21.56454	103	105	0.980952
22	63	M	5	S	-	160	72	28.125	112	108	1.037037
23	53	M	3	N	+	169	60	21.00767	86	99	0.868687
24	52	M	4	S	+	162	70	26.67276	98	104	0.942308
25	46	M	5	S	+	170	88	30.44983	116	118	0.983051
26	47	M	3	S	+	170	61	21.10727	88	96	0.916667
27	62	M	3	S	+	164	78	29.00059	110	118	0.932203
28	72	M	4	N	+	159	66	26.10656	94	98	0.959184
29	61	M	4	N	+	158	75	30.04326	98	106	0.924528
30	71	M	3	N	+	164	65	24.16716	92	108	0.851852

SL.NO	AGE	SEX	SES	LS	SMOKING	HEIGHT	WEIGHT	BMI	WAIST	HIP	WHR
31	75	M	4	S	+	160	85	33.20313	106	111	0.954955
32	65	M	3	S	+	158	65	26.03749	98	104	0.942308
33	73	M	4	N	+	163	72	27.09925	113	120	0.941667
34	63	M	3	S	+	156	65	26.7094	104	106	0.981132
35	63	M	2	S	+	160	70	27.34375	89	91	0.978022
36	41	M	2	S	-	168	70	24.80159	96	96	1
37	42	M	2	S	-	164	80	29.7442	96	102	0.941176
38	31	M	2	S	-	172	75	25.35154	99	102	0.970588
39	53	M	2	S	-	164	72	26.76978	92	99	0.929293
40	44	M	2	S	-	163	57	21.45357	81	89	0.910112
41	52	M	2	S	-	166	79	28.66889	103	103	1
42	31	M	2	N	-	170	64	22.14533	86	96	0.895833
43	36	M	3	N	+	168	75	26.57313	85	96	0.885417
44	40	M	3	N	+	167	75	26.89232	102	103	0.990291
45	58	M	3	N	+	154	61	25.72103	89	92	0.967391
46	65	M	3	S	-	154	64	26.986	100	112	0.892857
47	32	M	3	S	-	171	84	28.72679	96	102	0.941176
48	42	M	3	S	+	163	67	25.21736	84	93	0.903226
49	54	M	3	S	+	159	65	25.71101	104	108	0.962963
50	63	M	3	S	+	157	65	26.37024	104	109	0.954128
51	38	M	4	S	+	166	80	29.03179	106	101	1.049505
52	56	M	4	S	+	154	62	26.14269	96	96	1
53	69	M	4	N	-	163	60	22.58271	88	90	0.977778
54	39	M	4	N	-	173	80	26.72993	95	102	0.931373
55	34	M	4	N	+	163	70	26.34649	96	110	0.872727
56	46	M	5	N	-	162	72	27.43484	94	99	0.949495
57	37	M	5	S	-	154	50	21.08281	78	89	0.876404
58	73	M	5	S	-	165	72	26.44628	106	118	0.898305
59	72	F	4	N	-	146	60	28.14787	98	100	0.98
60	58	F	4	N	-	153	58	24.7768	80	92	0.869565

SL.NO	AGE	SEX	SES	LS	SMOKING	HEIGHT	WEIGHT	BMI	WAIST	HIP	WHR
61	57	F	4	N	-	153	71	30.33021	94	113	0.831858
62	40	F	4	N	-	160	97	37.89063	103	117	0.880342
63	67	F	4	N	-	146	50	23.45656	90	106	0.849057
64	50	F	4	N	-	163	57	21.45357	84	110	0.763636
65	65	F	4	N	-	152	57	24.67105	80	91	0.879121
66	65	F	4	S	-	153	58	24.7768	80	90	0.888889
67	67	F	4	S	-	146	58	27.20961	98	100	0.98
68	68	F	4	S	-	145	83	39.47681	107	120	0.891667
69	66	F	4	S	-	156	60	24.65483	86	100	0.86
70	37	F	4	S	-	157	55	22.31328	83	95	0.873684
71	41	F	4	S	-	160	56	21.875	85	105	0.809524
72	40	F	4	S	-	154	61	25.72103	87	106	0.820755
73	38	F	4	S	-	145	53	25.20809	82	96	0.854167
74	45	F	4	S	-	152	76	32.89474	101	122	0.827869
75	68	F	5	S	-	156	50	20.54569	86	100	0.86
76	59	F	5	N	-	167	65	23.30668	83	101	0.821782
77	74	F	5	N	-	149	50	22.52151	82	103	0.796117
78	32	F	5	S	-	147	55	25.45236	82	96	0.854167
79	48	F	5	S	-	139	64	33.12458	105	120	0.875
80	65	F	3	S	-	148	50	22.82688	83	96	0.864583
81	62	F	3	S	-	153	65	27.7671	88	95	0.926316
82	68	F	3	S	-	145	85	40.42806	108	127	0.850394
83	63	F	3	N	-	150	64	28.44444	85	83	1.024096
84	54	F	3	S	-	138	70	36.75698	106	118	0.898305
85	39	F	3	S	-	152	80	34.62604	100	108	0.925926
86	74	F	3	S	-	153	60	25.63117	89	101	0.881188
87	63	F	3	S	-	151	66	28.9461	98	100	0.98
88	52	F	3	S	-	150	65	28.88889	96	99	0.969697
89	30	F	3	S	-	153	65	27.7671	85	99	0.858586
90	55	F	3	N	-	145	85	40.42806	108	127	0.850394

SL.NO	AGE	SEX	SES	LS	SMOKING	HEIGHT	WEIGHT	BMI	WAIST	HIP	WHR
91	60	F	2	S	-	158	58	23.23346	87	101	0.861386
92	70	F	2	S	-	150	90	40	104	120	0.866667
93	67	F	2	S	-	150	90	40	102	123	0.829268
94	71	F	2	S	-	153	66	28.19428	100	101	0.990099
95	61	F	2	N	-	150	66	29.33333	98	98	1
96	69	F	2	N	-	160	52	20.3125	82	103	0.796117
97	70	F	2	N	-	153	60	25.63117	84	101	0.831683
98	52	F	2	S	-	150	90	40	102	123	0.829268
99	55	F	2	S	-	138	70	36.75698	106	124	0.854839
100	39	F	3	N	-	159	72	28.47989	99	101	0.980198
101	54	F	3	S	-	152	80	34.62604	100	120	0.833333
102	39	F	3	N	-	147	85	39.33546	82	96	0.854167
103	48	F	4	N	-	150	62	27.55556	89	106	0.839623
104	45	F	4	N	-	147	55	25.45236	83	96	0.864583
105	60	F	5	N	-	146	65	30.49353	95	103	0.92233
106	70	F	2	S	-	148	60	27.39226	87	109	0.798165
107	60	F	5	N	-	146	65	30.49353	95	103	0.92233

SL.NO	SBP	DBP	STAGE	FBS	PPBS	IFG/IGT	TC	HDL	LDL	VLDL	TG
1	148	96	1	97	132	N	163	30	101	32.2	156
2	166	80	2	99	158	Y	213	32	137	44.1	222
3	150	80	1	97	129	N	179	38	102	39	195
4	160	100	2	81	96	N	169	31	103	35.4	177
5	150	88	1	94	118	N	177	35	103	39	197
6	170	88	2	94	136	N	199	38	112	49	245
7	156	96	1	81	103	N	161	29	99	33.2	166
8	170	86	2	107	127	Y	200	33	135	42	210
9	180	88	2	113	141	Y	201	41	126	34	170
10	170	90	2	103	114	Y	179	28	102	49	250
11	164	90	2	107	154	Y	191	41	127	23	115
12	156	94	1	101	151	Y	185	41	124	20.2	101
13	162	86	2	107	136	Y	216	38	138	40	200
14	168	88	2	91	97	N	164	28	116	26.2	131
15	166	90	2	99	157	Y	204	38	130	36	180
16	156	80	1	103	131	N	183	31	120	32	160
17	162	90	2	91	107	N	181	30	121	30	151
18	150	80	1	96	128	N	160	29	100	31.2	156
19	160	80	2	86	114	N	191	38	104	49	245
20	164	86	2	102	144	Y	204	32	131	43.2	216
21	170	88	2	93	97	N	171	33	105	33.4	167
22	180	90	2	82	107	N	173	28	99	47	235
23	156	80	1	86	98	N	169	30	82	57	286.4
24	150	86	1	103	136	Y	264	44	183	37	186
25	170	90	2	96	117	N	217	40	105	72	302
26	164	80	2	114	142	Y	207	48	103	56	179
27	174	96	2	96	132	N	209	40	128	40.6	203
28	158	98	1	93	103	N	216	32	142	42	210
29	168	106	2	112	136	Y	220	40	133	47	235
30	156	86	1	81	93	N	216	30	140	46.2	231

SL.NO	SBP	DBP	STAGE	FBS	PPBS	IFG/IGT	TC	HDL	LDL	VLDL	TG
31	168	110	2	91	99	N	283	41	187	57	285
32	164	106	2	94	149	Y	268	40	169	59	295
33	156	90	1	78	113	N	216	30	138	48	240
34	144	100	2	88	98	N	244	36	171	37	185
35	150	70	1	104	145	Y	202	47	143	12	58
36	150	90	1	80	106	N	206	40	131	35	175
37	130	100	2	93	124	N	182	31	121	30	150
38	150	90	1	96	112	N	176	28	99	49	246
39	144	90	1	96	114	N	218	37	139	42	210
40	150	100	2	111	143	Y	237	40	154	43	215
41	150	90	1	96	130	N	206	38	133	35	175
42	160	110	2	96	110	N	188	41	124	23	115
43	160	100	2	86	117	N	188	34	119	45	225
44	180	120	2	88	119	N	179	36	112	31	155
45	160	100	2	108	162	Y	206	48	117	40.6	203
46	154	96	1	91	136	N	202	42	124	36	180
47	150	90	1	86	141	Y	205	33	130	42	209
48	150	110	2	120	176	Y	190	31	69	90	451
49	164	96	2	101	156	Y	192	43	77	72.2	361
50	160	90	2	110	158	Y	258	44	159	55	275
51	160	100	2	108	153	Y	198	45	111	42	212
52	190	140	2	109	122	Y	208	48	123.4	32.6	163
53	176	106	2	84	128	N	173	35	103	35.4	177
54	150	80	1	96	117	N	189	38	102	49	125
55	184	96	2	99	126	N	185	38	117	39.2	196
56	140	110	2	88	190	Y	152	28	98	26	130
57	144	96	1	94	126	N	162	29	100	33.2	166
58	180	86	2	101	146	Y	215	46	123	46.2	231
59	200	100	2	123	188	Y	184	38	108	37.2	188
60	156	88	1	96	107	N	175	46	104	29	145

SL.NO	SBP	DBP	STAGE	FBS	PPBS	IFG/IGT	TC	HDL	LDL	VLDL	TG
61	170	100	2	72	108	N	185	38	109	38	190
62	150	100	2	76	110	N	189	35	120	34	170
63	190	90	2	91	105	N	180	34	108	38	190
64	160	100	2	96	119	N	185	38	109	38	190
65	158	86	1	99	117	N	180	45	104	29	145
66	150	86	1	98	126	N	180	43	104	33	165
67	160	100	2	86	126	N	190	38	115	37.2	185
68	170	80	2	102	132	Y	207	45	126	36.4	182
69	168	106	2	98	120	N	179	29	107	43	215
70	176	90	2	88	120	N	170	33	105	32.2	161
71	148	86	1	92	115	N	175	40	104	31	155
72	148	80	1	80	117	N	175	40	104	31.4	157
73	158	98	1	90	120	N	183	47	101	34.1	171
74	160	98	2	98	146	Y	230	44	158	28	140
75	160	100	2	98	120	N	171	29	104	37.6	188
76	170	98	2	96	130	N	159	34	99	25.8	129
77	170	100	2	96	130	N	158	24	100	34	120
78	160	110	2	92	117	N	187	48	105	34	170
79	158	98	1	106	156	Y	208	44	64	99	495
80	180	90	2	98	120	N	174	33	109	32	160
81	172	98	2	88	102	N	196	45	118	33	166
82	150	90	1	97	105	N	205	44	127	34.4	172
83	170	106	2	98	140	Y	194	44	117	33	166
84	150	98	1	90	120	N	212	44	68	98.4	493
85	160	96	2	106	126	Y	235	45	159	31	155
86	190	80	2	90	128	N	216	42	123	51	255
87	160	90	2	98	107	N	206	40	130	36	180
88	160	90	2	96	103	N	207	41	130	36	180
89	170	110	2	96	112	N	195	45	117	33	166
90	150	90	1	96	106	N	206	45	126	34.4	172

SL.NO	SBP	DBP	STAGE	FBS	PPBS	IFG/IGT	TC	HDL	LDL	VLDL	TG
91	170	90	2	96	130	N	150	30	94	25.8	129
92	164	90	2	90	120	N	273	43	190	39.2	196
93	160	80	2	106	148	Y	253	33	190	29.2	146
94	192	80	2	90	132	N	220	44	125	51	255
95	164	86	2	88	96	N	208	39	133	36.8	184
96	170	106	2	98	140	Y	166	26	102	37.6	188
97	190	80	2	96	114	N	218	44	123	51	255
98	164	90	2	88	120	N	263	43	190	29.2	146
99	160	100	2	96	127	N	210	46	64	99	493
100	170	100	2	99	132	N	156	34	96	25.8	128
101	160	110	2	80	121	N	233	47	159	27	135
102	160	110	2	92	128	N	185	50	101	34	170
103	142	80	1	85	127	N	172	41	100	31	155
104	180	100	2	99	126	N	172	35	109	32	160
105	170	110	2	112	136	N	169	29	102	38	188
106	180	100	2	92	104	N	235	53	143	39	195
107	170	110	2	112	146	Y	169	29	102	37.6	188

MASTER CHART – NORMOTENSIVE CONTROLS NO - 60											
SL.NO	AGE	SEX	SES	LS	SMOKING	HEIGHT	WEIGHT	BMI	WAIST	HIP	WHR
1	65	M	3	S	N	164	66	24.53896	98	99	0.989899
2	39	M	3	S	N	168	60	21.2585	98	103	0.951456
3	54	M	3	N	N	166	82	29.75758	91	108	0.842593
4	40	M	3	S	N	157	60	24.34176	89	97	0.917526
5	41	M	3	S	N	164	75	27.88519	91	100	0.91
6	31	M	2	N	N	167	62	22.23099	80	89	0.898876
7	63	M	4	S	Y	164	60	22.30815	90	101	0.891089
8	61	M	4	S	N	172	60	20.28123	88	96	0.916667
9	66	M	5	N	N	159	57	22.54658	91	103	0.883495
10	61	M	3	S	Y	164	80	29.7442	93	110	0.845455
11	73	M	3	S	N	172	70	23.66144	93	99	0.939394
12	71	M	3	N	N	168	70	24.80159	96	99	0.969697
13	54	M	4	N	Y	160	58	22.65625	96	106	0.90566
14	44	M	4	S	Y	164	54	20.07733	96	102	0.941176
15	41	M	4	N	Y	168	74	26.21882	97	114	0.850877
16	62	M	4	N	N	184	58	17.13138	79	89	0.88764
17	33	M	4	N	N	168	73	25.86451	96	108	0.888889
18	62	M	4	N	N	174	62	20.47827	94	99	0.949495
19	60	M	4	N	Y	170	72	24.91349	94	106	0.886792
20	51	M	5	N	N	166	70	25.40282	96	110	0.872727
21	50	M	5	N	N	174	60	19.81768	92	100	0.92
22	60	M	5	N	N	168	70	24.80159	97	112	0.866071
23	69	M	3	S	N	162	60	22.86237	98	114	0.859649
24	54	M	4	N	N	156	54	22.18935	94	104	0.903846
25	52	M	5	N	N	174	61	20.14797	86	91	0.945055
26	53	M	5	S	Y	164	56	20.82094	90	103	0.873786
27	52	M	5	S	Y	156	54	22.18935	96	98	0.979592
28	64	M	3	S	Y	169	62	21.70792	82	88	0.931818
29	67	M	2	S	N	168	76	26.92744	98	104	0.942308
30	61	M	2	S	Y	168	70	24.80159	106	117	0.905983

SL.NO	AGE	SEX	SES	LS	SMOKING	HEIGHT	WEIGHT	BMI	WAIST	HIP	WHR
31	62	M	3	S	Y	182	60	18.11375	81	93	0.870968
32	46	M	3	S	Y	168	72	25.5102	90	102	0.882353
33	48	M	2	S	Y	172	70	23.66144	108	117	0.923077
34	68	F	5	N	N	170	68	23.52941	98	109	0.899083
35	74	F	5	N	N	169	69	24.15882	102	101	1.009901
36	48	F	5	N	N	160	68	26.5625	96	112	0.857143
37	69	F	3	S	N	170	63	21.79931	94	100	0.94
38	39	F	3	N	N	156	60	24.65483	80	94	0.851064
39	33	F	3	S	N	146	50	23.45656	88	96	0.916667
40	74	F	3	S	N	158	63	25.23634	84	98	0.857143
41	93	F	4	N	N	140	50	25.5102	85	96	0.885417
42	61	F	4	N	N	158	65	26.03749	98	102	0.960784
43	51	F	4	S	N	152	60	25.96953	80	92	0.869565
44	45	F	4	S	N	150	57	25.33333	88	98	0.897959
45	55	F	4	S	N	158	62	24.83576	84	99	0.848485
46	67	F	4	N	N	162	66	25.14861	108	119	0.907563
47	48	F	2	S	N	161	76	29.31986	112	118	0.949153
48	39	F	2	N	N	156	70	28.76397	91	103	0.883495
49	35	F	2	S	N	146	58	27.20961	86	106	0.811321
50	31	F	2	S	N	159	62	24.52435	84	98	0.857143
51	70	F	4	N	N	171	68	23.25502	101	114	0.885965
52	63	F	4	N	N	164	56	20.82094	88	98	0.897959
53	46	F	3	S	N	166	59	21.41094	96	98	0.979592
54	61	F	4	S	N	168	71	25.1559	94	107	0.878505
55	59	F	4	S	N	148	50	22.82688	84	117	0.717949
56	49	F	4	S	N	148	50	22.82688	85	96	0.885417
57	57	F	3	S	N	148	54	24.65303	82	88	0.931818
58	60	F	3	S	N	160	60	23.4375	83	100	0.83
59	65	F	3	N	N	162	62	23.62445	99	105	0.942857
60	63	F	4	N	N	156	70	28.76397	112	115	0.973913

SL.NO	SBP	DBP	STAGE	FBS	PPBS	IFG/IGT	TC	HDL	LDL	VLDL	TG
1	132	80	0	106	132	Y	169	41	115	13.2	66
2	120	80	0	86	98	N	168	42	106	29.2	146
3	130	80	0	94	98	N	176	56	10	13.4	67
4	120	80	0	77	110	N	154	39	87	28	140
5	120	80	0	98	106	N	166	29	110	27	135
6	110	70	0	89	97	N	177	37	102	38.6	193
7	128	86	0	92	114	N	172	59	93	20	100
8	130	82	0	86	107	N	137	37	86	14	68
9	116	80	0	87	114	N	156	41	89	26	130
10	136	80	0	96	104	N	169	56	99	13.4	68
11	122	86	0	94	120	N	166	39	104	23	115
12	122	80	0	93	199	Y	160	42	98	29.2	146
13	124	82	0	101	129	Y	162	39	100	23	116
14	120	80	0	90	114	N	164	40	104	29	145
15	120	80	0	84	119	N	166	29	104	33	164
16	120	80	0	89	112	N	160	44	94	21.6	108
17	120	86	0	96	98	N	134	41	81	12	60
18	124	84	0	99	114	N	127	41	68	18	90
19	124	80	0	90	90	N	130	40	80	10	40
20	124	82	0	90	106	N	160	48	93	19	95
21	118	78	0	79	89	N	129	41	67	21	105
22	122	84	0	89	99	N	138	41	83	15	75
23	120	80	0	102	132	Y	160	39	99	22	110
24	120	78	0	98	130	N	160	40	97	23.2	116
25	124	78	0	91	115	N	139	39	84	16	78
26	120	80	0	90	116	N	170	57	91	22	110
27	130	80	0	90	112	N	171	41	117	31	65
28	114	70	0	90	96	N	177	42	97	38	193
29	126	82	0	88	126	N	165	48	95	22	110
30	126	80	0	104	126	Y	180	48	104	28	140

SL.NO	SBP	DBP	STAGE	FBS	PPBS	IFG/IGT	TC	HDL	LDL	VLDL	TG
31	120	76	0	93	114	N	162	44	96	21	110
32	116	80	0	99	115	N	164	31	114	27	135
33	120	84	0	98	129	N	182	48	107	27	135
34	128	86	0	98	114	N	137	22	94	21.4	107
35	130	80	0	95	126	N	142	40	84	18	88
36	124	80	0	99	100	N	154	35	92	26.6	133
37	120	80	0	78	116	N	181	37	126	18.4	92
38	110	80	0	99	105	N	186	42	108	36	180
39	120	80	0	90	106	N	174	44	107	23.2	116
40	130	80	0	91	100	N	196	49	127	20.2	101
41	120	80	0	86	112	N	166	46	100	20	100
42	120	82	0	99	121	N	156	35	94	2608	134
43	122	82	0	98	128	N	172	41	100	31	155
44	122	80	0	92	112	N	172	35	104	33.2	166
45	110	70	0	98	115	N	176	42	98	36	180
46	136	86	0	102	146	Y	162	45	85	31.6	158
47	134	80	0	116	164	Y	244	36	171	37	185
48	130	80	0	106	154	Y	198	49	115	34.2	171
49	126	86	0	96	114	N	207	49	132	26	130
50	132	86	0	91	136	N	195	42	120	33	166
51	128	88	0	91	99	N	150	48	89	13	64
52	132	86	0	84	108	N	160	39	101	20	99
53	134	86	0	86	98	N	185	48	121	16	78
54	130	80	0	70	126	N	158	48	90	20	100
55	122	86	0	98	122	N	205	49	130	26	130
56	124	80	0	84	114	N	166	46	98	22	110
57	120	82	0	88	120	N	174	48	99	26.2	121
58	130	82	0	93	104	N	196	59	117	20.2	101
59	134	86	0	89	110	N	142	45	78	19	94
60	136	88	0	124	164	Y	129	21	52	55.8	279